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General Article

THE SOURCES OF SOME POISONS AND THEIR EFFECTS ON ANIMALS*

G. W. CLOUGH, D.SC.

(*late* PROFESSOR OF CHEMISTRY AND TOXICOLOGY, ROYAL VETERINARY COLLEGE, LONDON)

For convenience of description, poisons have been classified in various ways. Some domestic animals are poisoned wilfully and others accidentally. The former cases are exceedingly small in number in comparison with the latter. There is an erroneous impression in the minds of many owners of dogs and cats that large numbers of these animals are poisoned wilfully and when their pets become suddenly ill or die, they assert that the animals have been wilfully poisoned. This attitude is encouraged by the occasional headlines of the daily press. When show horses, sheep in a public park or other animals have died suddenly, the owners have at once suspected that fellow competitors, ill-disposed persons, servants of the owners or mysterious strangers have wilfully poisoned the animals, and they have communicated their suspicions to representatives of newspapers. Although there are occasionally cases of the wilful poisoning of animals in this country, they are comparatively rare and the publication in the press of suspicions without any proof should be discouraged. For the purpose of this address I propose to deal with some poisons injurious to animals according to the sources of the poisonous substances; some of these may therefore occur in more than one class, but this will not prove to be a serious disadvantage in the discussion.

INDUSTRIAL POISONS

There are numerous poisonous substances which are produced or used in industrial processes. Some of these are emitted as gases, vapours or dust into the atmosphere and in the presence of moisture they may be subsequently deposited on pasturage. Others are waste-products which may be dumped on the ground or discharged as effluents into streams. It has been stated by competent authorities that Great Britain has yielded mineral treasure for the use of man for at least three thousand years and that most probably the earliest evidence of mining in Europe is to be found in Cornwall. Since the working of some of the minerals must have been carried on near agricultural land, it appears inevitable that there have been in the past cases of poisoning in animals in Cornwall and in other parts of Great Britain by materials emanating from the mines and their workings. In the roasting of ores containing sulphur, large quantities of *sulphur dioxide* and some *arsenious oxide* are produced and ultimately *sulphuric acid* and the arsenic are found on vegetation. There are accounts of chronic arsenical poisoning in animals from this cause in English and in Continental literature, but sufficient attention does not appear to have been directed to the possible effects on animals of sulphur dioxide in the atmosphere and sulphuric acid on the herbage and in the soil. The former may be responsible for respiratory symptoms and in the light of present knowledge of deficiency diseases, it is not improbable that the washing out of lime and other minerals from the soil by sulphuric acid resulted in the coarse grass growing on such soil being deficient in some of those minerals which are essential for the health of farm animals. Lyon Playfair, in 1862, stated that in copper smelting sulphur dioxide goes into the atmosphere and that "sulphuric acid vapour forms a heavy cloud and descends readily to the ground. This vapour is highly injurious to vegetable and affects animal life. The cattle fed on the grass in the neighbourhood of these works get a thickening of the joints" (*Evidence before Select Committee of the House of Lords on Injury from Noxious Fumes*, 1862). *Hydrogen sulphide* is a very poisonous compound and considerable damage was caused by this substance in Suffolk a few years ago; the source was an artificial silk factory, the effluent from which was allowed to flow into the River Gipping. When effluents containing large quantities of vegetable waste are discharged into sluggish streams, fermentation may take place with the production of much sulphuretted hydrogen from the proteins containing sulphur. The effluents from fruit canning operations have been responsible for the deaths of poultry; it is very probable that hydrogen sulphide was the

SULPHUR DIOXIDE

SULPHURIC ACID

HYDROGEN SULPHIDE

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cause of the damage. In some manufactures *hydrofluoric acid* is emitted, a vapour very soluble in water, and in the presence of atmospheric moisture it is deposited on pastures in the neighbourhood of such works. It has been reported that pastures adjoining aluminium factories in which cryolite is worked have been found to contain fluorine compounds. Animals fed on such pastures have exhibited symptoms of anæmia, emaciation, stiffness and a tendency to fracture of the bones. The bone lesions resemble those of osteomalacia (*Norsk. Vet. Tidskr.*, 1934, **46**, 61). Hupke and Götze have described an illness of cattle near a chemical factory; the symptoms were lameness, sore feet, swellings round the pastern joints. The condition was ascribed to osteomalacia; investigation revealed that the disease was caused by fluorine compounds. They also describe how cattle were affected by fumes containing fluorine compounds originating from a phosphate factory (*Deuts. tierärztl. Wschr.*, 1931, **39**, 203). Hydrofluoric acid in the atmosphere may cause respiratory irritation. Miessner considers that calcium is withdrawn from the bones of animals feeding on herbage contaminated with hydrofluoric acid emanating from artificial fertiliser factories.

Hydrofluoric Acid

The increasing use of *cyanides* in metallurgical operations (*e.g.*, metal plating) and in fumigation for the destruction of rabbits, rats and insect pests has led to some cases of cyanide poisoning in domestic animals (*Vet. Rec.*, 1933, **13**, 538). A cow was found dead and a day later four more cows and six ducks were also found dead. Two samples of stream water to which the animals had access contained cyanides to the extent of 10 grains of hydrocyanic acid per gallon. Copper, chromium and nickel compounds were also present in the water. The cyanide was traced to an effluent from a chromium plating works situated two miles from the farm on which the animals died. A cow was discovered ill with symptoms of champing and staggering; it died within one hour. Hydrocyanic acid ($\frac{2}{100}$ part of a grain per pound) was found in the rumen contents. A sample of surface water from which the animal had been drinking contained $\frac{1}{3}$ grain hydrocyanic acid per gallon and a sample of stream water $\frac{1}{10}$ grain per gallon. The cyanide was believed to originate from timber works.

Cyanides

The washings from arsenical minerals may contain *arsenic* compounds and the beds of streams into which the washings flow may become comparatively rich in arsenic. These compounds of arsenic are insoluble in water, but if they are taken by cattle and rendered soluble by the gastric fluids, symptoms of chronic poisoning may occur. It may be recalled that such an insoluble compound as iron oxide has proved to be invaluable as an addition to food for animals in areas deficient in that mineral. It must, however, be pointed out that all specimens of iron oxide are not equally effective; this is doubtless due to the varying rates of solubility of the specimens in acids. Arsenical poisoning in horses and cattle feeding on grass in the neighbourhood of a chemical factory recently occurred in Essex. Twenty bullocks had been on marshes for five weeks; six exhibited symptoms of watery diarrhœa and rapid wasting followed by collapse and death, whilst the remainder were in poor condition. Arsenic was found in the stomach contents; two samples of grass contained half a grain and a quarter of a grain of arsenic per pound of grass; a sample of drinking water from the marshes contained ten grains of arsenic per gallon. A short time afterwards hair from three Shire horses in the same district was submitted for analysis; arsenic was present to the extent of $\frac{1}{4}$, $\frac{2}{5}$ and $\frac{2}{5}$ grain per ounce in the washed hair. The symptoms exhibited by these horses were pain, thirst and tucked-up appearance of abdomen. On removal from the field they became normal in three weeks.

Arsenic

Pastures may become contaminated with *lead* compounds from mines by water carrying down lead which ultimately settles on the fields or by wind blowing dust from refuse heaps on to the fields. Lead sulphate is emitted in the smoke from lead smelters and may be deposited on vegetation up to a mile from the works. Lead ores occurring in the carboniferous limestone of the Pennines between Northumberland and Derbyshire were mined and worked by the Romans; lead smelting has also been carried on in the Mendip Hills and in Wales. In 1853 lead smelting on the Mendip Hills was followed by lead poisoning in cattle, lambs and colts. It was in connection with one of these cases that Herepath stated that there was a blue line on the gum of a cow's lower jaw, that he dissected out this line and reduced it to metallic lead; the metal was exhibited to the jury in an action for damages. Lead poisoning in anima's as a result of lead smelting is at present rare, if it exists at all, in this country. This is due partly to the fact that lead smelting in Great Britain is an almost extinct industry and partly to the improvements in the operations, which have much diminished the loss of lead compounds from the flues. Moreover, lead sulphate is one of the most sparingly soluble and least toxic compounds of lead. In this type of poisoning the chief injury produced has been observed in horses; the lungs were affected making breathing difficult and

Lead

the animals often became " roarers." A survey of livestock was made in 1914 in the Selby smoke zone in California. Lead on vegetation was found to be 21 parts per million (=13 grains per 100 lb.). The conclusions drawn from the survey were: there was no evidence of suspicion that the smelter smoke had ever been injurious to cattle or hogs, but that it appeared probable that in the past considerable damage to horses from lead poisoning had occurred in the smoke zone from the Selby smelter; there was no evidence to believe that at the time of the survey the smelter smoke was doing any injury to livestock in the Selby smelter zone (*Report of the Selby Smelter Commission*, 1915, 502). Such an enquiry as that carried out by this Commission seems to be the only satisfactory way of dealing with legal controversies concerning the damage inflicted by the mining and metallurgical industries. Dunn and Bloxam found that lead was present in the herbage and soil of lands adjoining certain coke ovens and concluded that the illness and deaths of cattle and sheep fed thereon were due to this cause. They stated that on a farm in proximity to a coke oven 30 animals had died and that others exhibited symptoms of illness. Lead was found on the grass of two fields in amounts of 14 and 11·5 parts per million (= 10 and 8 grains per 100 lb.). The organs of animals were said to present the *post-mortem* aspects of death by lead poisoning. Lead was found in the stomach tissue of a sheep to the extent of 0·6 parts per million (= $\frac{1}{40000}$th grain per ounce) and in the liver the amount was 2·6 per million (= $\frac{1}{9000}$th grain per ounce) (*J.S.C.I.*, 1932, **51**, 100T). I expressed the opinion that in the absence of reports of reliable clinical inspections and of *post-mortem* examinations the evidence advanced by Dunn and Bloxam in their paper was quite inadequate to prove either that the illness and deaths of any animals had been due to lead poisoning or that the pastures had been " poisoned " with lead compounds (*Chemistry and Industry*, 1932, **51**, 526).

Salt A salt works effluent containing a high proportion of *common salt* has been proved to be very deleterious to cattle (Scott, *V.J.*, 1924, **80**, 19).

Cement-Works Fumes From time to time there have been complaints from farmers that their crops and livestock have been damaged by fumes emanating from cement works. In some cases damage to fruit trees and other vegetation has been conclusively proved; sufficiently thorough investigations have not been made to enable a definite conclusion to be drawn concerning the alleged damage to animals from this cause.

Tar Acids Tar acids discharged into rivers from coke oven and other effluents have been suspected of causing poisoning in cattle and in one legal action damages were awarded for the loss of a cow from this cause. The amounts of tar acids present in such rivers are, however, so small compared with lethal doses that there is no justification for the belief that they cause ill effects in cattle.

LEAD AND ITS COMMON TOXIC COMPOUNDS

Metallic Lead Birds are the animals most susceptible to poisoning by *metallic lead*. *The Field* in 1876 and 1882 directed attention to losses of pheasants owing to the birds picking up lead shot on extensive shooting preserves in England. Wetmore has found that six pellets of No. 6 shot constitute a fatal amount of lead for a wild mallard and in some cases three shot were sufficient to cause death (*U.S. Dept. of Agric.*, 1919, *Bulletin* 793). In 1921 thousands of water fowl died in Louisiana as a result of picking up lead shot in areas frequented by sportsmen. In Great Britain there are far greater losses amongst cattle from acute lead than from any other form of poisoning. The sources of the lead are usually paint, *white lead* or *red lead*. Cattle will lick paint from their boxes; this may be old paint, there may be no trouble for years and then cattle, especially calves, will remove the paint with fatal consequences. Deaths of cattle have recently occurred after they had eaten red lead which had been left on fields by contractors working on the electricity scheme or on drainage schemes; there have also been numerous fatalities due to lead poisoning in the vicinity of new housing estates. In some of the latter cases antimony as well as lead has been found in the viscera; both of these were doubtless present in paint which had been left in places within reach of the animals. Acute lead poisoning in the dog is illustrated by the following recent example. A one-year-old terrier dog became dull, refused food, vomited and had violent attacks of shivering and grunting; these symptoms were followed by weakness in the hind quarters, frequent fits, frenzy, delirium and partial blindness. It died two days after the onset of symptoms. Lead was found in stomach wall and liver. The origin of the lead was paint. The electric batteries so extensively used in radio receivers and motor cars constitute another source of lead which may be the cause of poisoning. Some beasts became ill after grazing in a park; the temperature was subnormal, pulse thin and rapid. The animals exhibited intense depression and passed small amounts of hard fæces. Death in convulsions followed 12 to 24 hours after the onset of symp-

toms. The *post-mortem* examination revealed inflammation and impaction of the omasum, congestion of liver and kidneys. Lead ($\frac{1}{10}$th grain per ounce) was separated from the ingesta of a bullock. A tank to which the animals had had access and in which some batteries had been washed out was found to contain a quantity of metallic lead and lead oxide.

Lead Arsenate *Lead arsenate*, an insecticide employed for spraying fruit trees, is a very poisonous compound. Witter is of opinion that in America it is one of the most common causes of lead poisoning in domestic animals and mentions a case in which six cows died and several others were very seriously ill; the cause of the damage was conclusively proved to be lead arsenate (*Vet. Med.*, 1934, **29**, 512).

WEED KILLERS

Sodium Arsenite Solutions of sodium arsenite, ammonium sulphate, copper sulphate, ferrous sulphate, ammonium sulphocyanide, sodium carbonate and sodium chlorate have been employed for the destruction of weeds. One of the commonest vehicles of arsenic in cases of arsenical poisoning amongst animals is arsenical weed killer. The menace to animals of this material is exemplified by the following recent cases. A farmer sprayed a field with weed killer to destroy docks. Two cows were turned out into the field and in 24 hours they exhibited typical symptoms of acute arsenical poisoning. The diagnosis was confirmed by a chemical analysis of the viscera of one of the animals. Ten cows entered a kitchen garden containing long grass on which weed killer had been sprayed a few days previously. Four died and the others were ill. Arsenic was found in the stomach contents of one to the extent of $\frac{1}{30}$th grain per ounce. A cow exhibited symptoms of pain, purging, grinding of teeth and salivation; it died in 12 hours. Arsenic was found in the ingesta. Nettles in the field had been watered with weed killer. A Scotch terrier was normal in the morning but later there was vomiting and diarrhœa; death followed in 12 hours. The liver and intestines contained arsenic. Weed killer had been used on the premises the day before the dog's illness. The most effective antidote in arsenical (or mercurial) poisoning is sodium thiosulphate administered intravenously. For example, 10 c.c. of a 5 per cent. solution may be used for the dog; this may be repeated in five hours and again 12 hours later. Some years ago, when discussing the question of arsenical poisoning by weed killer, insecticides and sheep dip, I remarked that "arsenic is by no means indispensable in horticulture or agriculture and there would be considerable saving of animal life if its use were restricted" (*Vet. Rec.*, 1927, **7**, 209). In *The Report of the Poisons Board* (1935) arsenic oxides and sulphides, sodium arsenite, arsenate and thioarsenate are placed in the Fourth Schedule to the Rules and no listed seller may sell these except for use as sheep dips or sheep washes. It will, therefore, be necessary to employ only non-arsenical substances as weed killers. Of these, washing soda is very useful, but sodium chlorate is, in general, the most effective. Investigations have been made in America, Australia and New Zealand on the value of sodium chlorate for the destruction of such weeds as ragwort. Clouston and Hill recommend spraying with a $2\frac{1}{2}$ per cent. solution for the eradication of chickweed, daisy, common nettle and sheep's sorrel. Stewart and Wright found that a dilute solution of potassium chlorate quickly destroyed Ranunculaceæ, nettles and ground ivy (*Univ. of Cambridge, Inst. An. Path. Rep.*, 1932-33). The fatal doses of sodium arsenite for farm animals have been determined by Green and Dijkman in S. Africa and by Seddon in Australia, whilst those of the chlorates for some animals have been published by Seddon and McGrath, Fröhner, Zimmerman and also by Steyn (*Onderstepoort Journ.*, 1933, **1**, 157).

Approximate Lethal Doses of Arsenious Oxide (in the Form of Sodium Arsenite), Sodium Chlorate and Potassium Chlorate

	Arsenious Oxide grains	Sodium Chlorate ounces	Potassium Chlorate ounces
Horse ...	30-45	5	9
Cow	30-60	10	18
Sheep ...	6-10	2	4
Dog	5	—	2

Evidently sodium chlorate is a compound of comparatively low toxicity and there is much less risk of damage to animals by weed killer containing this substance than by one containing sodium arsenite. The chief disadvantage of sodium chlorate is the risk of fire when material on which it has been sprayed becomes dry. A spark may then cause conflagration owing to the rapidity of oxidation of organic matter by chlorates. It is obvious that those using the substance should avoid spilling it on their clothes.

SHEEP DIPS

The common sheep dips are the sulphur, arsenical and carbolic dips. The chief advantage of arsenical dips over sulphur (polysulphide) dips lies in the greater ease of preparation of the former; still more easily prepared are the so-called carbolic dips which have only to be diluted with the requisite quantity of moderately soft water. It is not without

interest to contrast the care with which druggists, doctors and veterinary surgeons keep their little bottles of arsenical preparations under lock and key with the careless way in which some farmers allow pounds of arsenical materials to lie about their premises and with the way in which large quantities of arsenic have been scattered about by the accidental breakage of large containers in docks, in railway sidings and on lorries. There is little danger to sheep in this country by dipping with arsenical dips if the well-known precautions are taken, but accidents sometimes arise owing to the animals swallowing the fluid or eating grass contaminated by the material. In South Africa arsenical preparations are in common use in agriculture and Van Zyl estimates that the cases of suspected arsenical poisoning probably run into several thousands annually (*Union of S. Africa Dept. of Agric., Ann Rep.*, 1929, 1189).

Arsenic

The following cases of poisoning by *arsenic*, the vehicle of which was sheep dip, have been encountered during the last few years. Calves gained access to a shed in which was a sack containing sheep dip. Four died after showing symptoms of acute diarrhœa, frothing and thirst. Arsenic was present in stomach contents and liver. A cow appeared normal 12 hours before it was found dead in a field. It was reported that a yellow dye had got on to the field as a result of a collision in which barrels of the material burst and spread over the ground; this had burnt the grass. Arsenic was found in the cow's stomach contents. It is very probable that the material was not a dye but was arsenical sheep dip. A farmer (A) dipped his sheep in his neighbour's yard, which was was separated from A's farm by a railway. When he had completed the dipping, A cleaned out the bath and poured the surplus dip down a drain; this drain passed under the railway and emptied into a ditch on A's farm. When this ditch overflowed on to A's pastures, an outbreak of arsenical poisoning occurred amongst his cattle. In some counties sheep dip is used as a wash for killing lice on cattle. A farmer accidentally prepared too concentrated a solution of a liquid arsenical sheep dip and applied it to the necks of 28 cattle, ten of which died of acute arsenical poisoning. Chemical analysis of the skin and viscera indicated that the poison had been absorbed through the skin. Fatal accidents sometimes follow the use of the *carbolic dips*. In 1930 at the Kent Assizes, an action was brought in which £2,250 damages were claimed for the deaths of pedigree sheep after dipping with a carbolic dip (*Quested* v. *Morris Little & Sons*). A leaflet stated that the dip was "non-poisonous when used according to printed instructions." On one farm out of 12 pedigree rams and 50 lambs, nine rams and seven lambs died and on another of plaintiff's farms out of 12 rams and 20 lambs, four rams and three lambs died. There were no losses amongst clipped lambs and plaintiff contended that the dip was unsuitable for long-woolled sheep. Plaintiff's veterinary surgeons expressed the view that the damage was due to the poison being absorbed through the skin, but veterinary experts for the defence were of opinion that the symptoms were not consistent with absorption through the skin and that the animals died from inhalation pneumonia. Judgment was given for defendants. Owing to fatalities amongst sheep with certain proprietary carbolic dips in S. Africa, De Kock and Steyn carried out an experimental investigation. In their experiments the heads of one group of sheep were immersed, whereas in another group the heads were kept above the fluid to exclude every possibility of the animals swallowing the dipping liquid. There was heavy mortality (60 per cent.) in both groups of sheep. These experiments thus rendered definite proof that the animals were poisoned by absorption of the dipping fluid through the intact skin. The symptoms exhibited were respiratory distress, discharge from nose and marked salivation; the visible mucous membranes were cyanotic. The symptoms increased in severity, the animals became comatose, there was complete paralysis and unconsciousness when death took place 15 hours to a few days after the onset of symptoms. The *post-mortem* examinations revealed that the lungs were extremely affected in all cases. The authors assume that the cause of death in these cases was phenol poisoning, but it must be pointed out that there were probably compounds other than phenols in these dips, namely, tar hydrocarbons and tar bases—pyridine and allied compounds—(*S. Africa Dept. of Agric., Ann. Rep.*, 1929, 643). Salusbury has reported that a bull died after being dressed with a gallon of a 4 per cent. solution of Jeyes' fluid. He suspected that phenol poisoning was the cause of death; *post-mortem* examination revealed nothing unusual in the lungs and pleuræ (*Vet. Rec.*, 1934, **14**, 936). A mare recovered in ten days after receiving a pint of undiluted lysol (Shawcross, *Vet. Rec.*, 1933, **13**, 92). Since lysol contains about 50 per cent. of cresols, it would appear that this animal received about ten fluid ounces of cresols without a fatal result. Green found that a lamb (43 lb.) died after being given 100 c.c. of 5 per cent. lysol, *i.e.*, approximately 40 grains of cresols.

Phenol

Cattle have died from arsenical poisoning

after drinking from a trough which had contained an *arsenical dressing for foot rot* in sheep. A mysterious case of arsenical poisoning in bullocks was explained by the fact that the animals had grazed on long grass in a field into which sheep had been turned out after having walked through a foot bath containing arsenical dressing for foot rot.

WARBLE-FLY DRESSINGS

Deaths have sometimes followed the dressing of cattle by certain proprietary preparations employed against warble-fly. Five heifers were each dressed with less than half a pint of a preparation; they quickly showed symptoms of shivering, difficult respiration, foaming, groaning and paralysis. They fell to the ground, one died in half an hour and all were dead within two hours of the application of the dressing. The mucous membranes were blue and the lungs were acutely affected. Cattle which had not been dressed remained normal. The preparation was found to consist of hydrocarbon oils, *Dippel's animal oil* (*oleum animalis*) with a small quantity of pyrethrum extract.

I attributed the damage to the toxic action of the pyridine bases present in the animal oil. Several animals were dressed with another proprietary liquid against warble-fly. A young bull was found dead in a few hours. On the following morning a cow was down and unable to rise; there was a foam of saliva round the mouth, temperature was subnormal, the animal showed progressive paralysis and acute tympany; death occurred 12 hours later. *Post-mortem* examination revealed inflammation of the large intestine and cyanotic membranes. The preparation consisted of hydrocarbon oils and Dippel's oil (compare *Vet. Rec.*, 1925, **5**, 720). Whilst Derris root is not without toxic properties, it appears to be the safest material to use against warble-fly. In France a preparation containing paradichlorobenzene (a compound of very low toxicity for domestic animals) has been employed for the same purpose.

WORM KILLER

MOWRAH SEED

Mowrah Seed (*Bassia longifolia*) is often used on lawns and golf greens for the destruction of worms. The material which is sold for this purpose is the ground residue after the extraction of the fat from the seeds; it is applied to the greens, which are afterwards watered. The toxic principle is a sapo-glucoside, mowrin, which is said to have a digitalis-like action and has been investigated by Moore (*Biochem. Journ.*, 1911, **5**, 94). The worm killer may, however, give rise to blisters if handled without care. Cows grazing on a golf course which had been dressed with the material became very ill, the prominent symptoms being intense diarrhœa and paralysis; the illness lasted for about a week and was followed by recovery. In another district wild rabbits near a golf green which had been dressed with the seed became thin and many died; some were observed to stagger, fall over and die. The *post-mortem* revealed nothing definite except emaciation.

VERMIN KILLERS

The commonest poisons employed for the destruction of rats and other vermin are strychnine, phosphorus, barium carbonate, thallium sulphate, sodium fluoride, sodium fluosilicate, arsenic and red squill.

For many years the highest mortality due to poisoning in dogs in this country has been caused by *strychnine*, which has been put down in baits for killing rats or foxes. The Poisons Board recommend that the retail sale of strychnine should be prohibited except as an ingredient in a medicine and they point not only to its use as an instrument of murder but to the frequent poisoning of hounds, domestic animals and wild birds from the negligent laying down of poisoned baits for vermin (*Report*, 1935, 30).

STRYCHNINE

BARIUM CARBONATE

Barium carbonate is a deadly poison and cases of poisoning caused by this substance have been encountered in dogs and poultry.

PHOSPHORUS

Phosphorus is very frequently the cause of poisoning in poultry; dogs sometimes die as the result of eating baits containing this element, but these animals usually refuse to eat such baits.

THALLIUM

Thallium sulphate is another very deadly poison for domestic animals. It has been extensively employed in Germany and America for rat destruction. Zelio corn and zelio paste contain 2 to 3 per cent. of thallium. Roche Lynch and Scovell have discussed the toxicity of thallium and conclude that it is a highly toxic element showing a marked similarity to lead in its chemistry and its toxic properties. They are of opinion that it is not justifiable to use such a powerful poison as thallium acetate in the treatment of ringworm of the scalp (*Lancet*, 1930, **219**, 1340). The symptoms of thallium poisoning in dogs are vomition, refusal of food, depression, salivation, reddening of ears, loss of hair, excretion of blood through rectum; death occurs in from seven to 21 days. Munch has given a comprehensive review of the toxicity of thallium sulphate (*Journ. Amer. Phar. Assoc.*, 1928, **17**, 1086). The experimental investigations of Newsom, Loftus and Ward made in 1930 indicated that sheep do not readily eat oats mixed with thallium sulphate but that the salt is

poisonous to these animals when taken by the mouth. The lethal dose is about the same as for other animals, the minimum being 11 milligrammes of thallium per lb. body-weight; the least dose that caused the shedding of wool was 4 milligrammes per lb. body-weight. No effective antidote appears to have been discovered for this poison. Feeding experiments on cattle with *sodium fluoride* and *sodium fluosilicate* have demonstrated that 50 to 100 grains of these salts cause acute poisoning, whilst 30 to 45 grains given daily produce a gradual emaciation. The death of a horse has been ascribed to the eating of maize flour mixed with sodium fluosilicate (*Schweiz. Arch. Tierhlk.*, 1931, **73**, 237). In accordance with the Report of the Poisons Board (1935) it will be illegal to sell any rat poison containing a scheduled poison other than barium carbonate.

Sodium Fluoride

Sodium Fluosilicate

Red Squill is not a scheduled poison. This is a large onion-like bulb (*Urginea Maritima*) growing in South Italy, Sicily, Dalmatia and Morocco. Whilst rats readily consume baits containing red squill, other animals usually refuse to eat the material. The chief obstacle to this being an entirely satisfactory commercial rat poison lies in the difficulty of preparing uniformly toxic preparations; standardisation requires biological testing. Although many chemical studies have been made on the constituents of red squill, the active toxic principle has not been definitely isolated and identified. The chief symptoms of illness it produces are paralysis of the hind limbs, depression and convulsions. Rats die on account of respiratory failure. Winton states that the cardiac glucoside and the rat-poisoning principle of red squill are distinct substances. The former occurs in about equal amounts in red and white squills; the rat-poisoning substance is present in red squills only (*Journ. Pharmacol. and Expt. Ther.*, 1927, **31**, 123, 137). Munch, Silver and Horn have shown that red squill is toxic to rats, whereas white squill is not (*U.S. Dept. of Agric., Technical Bulletin*, No. 134, 1929). In their monograph these authors state that the powders obtained by drying the bulbs are usually more toxic than other preparations. The lethal dose of such powders is approximately 100 milligrams per lb. of body-weight of white rats. Cats, dogs, chickens and pigeons were not seriously harmed by squill powder, the material being either not eaten or, if eaten, vomited. I have prepared an extract of red squill by boiling the bulbs with water, cooling and adding salicylic acid as a preservative. This extract on bread was effective in destroying the rats at a factory near a canal in the East End of London. A few cases of suspected poisoning in dogs by red squill have been observed. The symptoms exhibited in a Cairn terrier bitch indicated the probability of squill poisoning. The animal vomited and in three hours became comatose; breathing was shallow and rapid, the pulse scarcely perceptible and occasional convulsive movements were noted. The dog died quietly after about six hours' illness. *Postmortem* examination revealed some patchy inflammation of the stomach and congestion of the vessels of the mesentery. The liver was friable and there were small hæmorrhages in the mucous membranes of the larynx and pharynx. Red squill had been used on the premises for killing rats. A comparison of the toxicities to domestic animals of the various rat poisons indicates that red squill conforms best with the requirements of a satisfactory rat poison with regard to safety and effectiveness. It cannot, however, be asserted that it is non-poisonous to animals other than rats.

Red Squill

DRUGS

Proprietary Drugs.—Many of the proprietary drugs issued by well-established firms possess genuine merit, but there are on the market numerous remedies for animal ailments which are of no value for the maladies which they are specified to cure. They sometimes consist of drugs of the pharmacopœia sold under special names; in some instances the patent medicine vendor exploits the mysteries of vitamins and radioactivity for selling mixtures containing aperient, astringent or stimulant drugs. On the other hand, a tribute must be paid to those business houses of repute which not only supply drugs of a high degree of purity and of tested and proved therapeutic action but which have also at considerable expense supplied a deficiency in medical and veterinary science, namely, pharmacological research. The veterinary profession is under an obligation to such firms on account of the many valuable researches which have been prosecuted by their staffs. I need only refer here to the investigations made at the Wellcome Research Laboratories and to the scientific papers in *Medizin und Chemie* published by the I. G. Farbenindustrie last year.

Many English agriculturists pay large sums annually for materials represented to be remedies or preventives of animal diseases; thus an extensively sold "abortion remedy" has been found to consist of brown sugar and wheat offal; another was a mixture of coal tar creso's and water. Poultry breeders frequently pay excessive prices for mixtures of common drugs which cannot possess the virtues enumerated in the advertisements. In 1906 the Congress of the United States passed the Federal Food and

Drugs Act and in 1910 the Federal Insecticides Act. These Acts protect the public from the fraudulent claims of the therapeutic efficiency of drugs and enable people to obtain proprietary preparations of drugs with the assurance that the claims made on the labels can be substantiated. The result has been that the majority of "abortion remedies" and many other proprietary mixtures have been removed from the American market (Woodward, *Address to Amer. Vet. Med. Ass.*, Aug., 1928). The Poisons Board in the section of their Report dealing with animal and poultry medicines state that "it would appear that there is a considerable retail sale of animal medicines containing poisons to farmers, cattle and horse breeders, poultry keepers and private owners of horses, dogs, cats and poultry by persons who are not lawfully 'keeping open shop.' Much of the trade is in the hands of manufacturing firms who deal direct with the animal owner or poultry keeper" (p. 10). Whilst such sales appear to have been illegal, the recommendations of the Board will legalise the sales of certain specified poisons, including carbon tetrachloride and oil of chenopodium. It may be useful to discuss the toxicity of carbon tetrachloride and other *simple organic chlorine compounds*.

Methyl chloride

Methyl chloride, CH_3Cl, is an odourless gas at atmospheric pressure. It possesses some narcotic action but exerts a toxic action on the nervous system. Its employment in domestic refrigerators has caused much illness and many deaths (*B.M.J.*, 1929, 633). In Chicago alone in 1928-29 there were 29 cases of methyl chloride poisoning and of these ten were fatal.

Methylene chloride

Methylene chloride, CH_2Cl_2, is a liquid which possesses narcotic action; the Bayer Company have introduced it in a chemically pure state under the name Solæsthin. Its toxicity appears to be low.

Chloroform

The merits and dangers of *chloroform*, $CHCl_3$, are well known and need not be described here:

Carbon tetrachloride

Carbon tetrachloride, CCl_4, is extensively employed as a solvent for oils and fats, as a dry cleaner and as a fire extinextinguisher. In medicine it has been much used against hookworm and also for liver fluke. Hall and Shillinger gave sheep up to 48 c.c. without ill effects; Sheather gave a lamb 50 c.c. in liquid paraffin without a fatal result; Daubney has given a sheep (79 lb.) 45 c.c. without fatality and states that sheep may be given extremely large doses of the drug with complete safety when magnesium sulphate solution is also administered (*V.J.*, 1930, **86**, 5). Daubney thus confirms the results of Hall and Shillinger who found that magnesium sulphate reduces the toxicity of the drug to dogs. In the treatment of liver fluke the dose commonly given to sheep is 1 c.c. in a capsule and from the above results it would be anticipated that this amount would be a perfectly safe dose. Unfortunately this quantity has proved fatal to a considerable number of sheep in this country. It must, however, be pointed out that the fatalities constitute a very small proportion of animals which have been treated with this compound. It has been repeatedly emphasised by writers that the drug should be free from impurities and that the toxic action may be due to the presence of impurities in the material employed. The manufacture of chemically pure carbon tetrachloride is carried on in England on a large scale by a reputable firm and in my opinion the compound sold by English wholesale houses is beyond suspicion with regard to purity.

I may mention a few recent accidents with this drug.

One hundred ewes in good condition were each dosed with 1 c.c. On the next morning about half of the animals were ill, their ears down and abdomens tucked up. Three died and the remainder continued in a poor condition for some weeks; a number of them aborted. Eleven sheep died out of 200 which were dosed (1 c.c.); many others refused food and had a staggering gait. Eighty sheep were each given 1 c.c. One hour after dosing two were observed to be ill; six died. *Post-mortem* examination on one showed septic pneumonia, acute inflammation of the trachea and larynx and necrosis of adjacent tissues. Evidently in this case some of the capsules were broken during the administration of the drug which entered the trachea. The damage was due to inhalation intoxication following faulty administration of the drug. Carbon tetrachloride is not well tolerated by cattle and it is inadvisable to employ it for these animals. Sixteen bullocks were each given 10 c.c. Three died on the second and two on the third day after administration. Six others were ill with symptoms of pain and staggering gait. *Post-mortem* examination revealed some peri-endocarditis, enteritis with petechiæ on the large bowel, blood-stained fæces and on the livers were yellow patches extending to a depth of ¼ inch to 1 inch. Two bullocks each received 4 c.c. and showed signs of distress within 12 hours. They died in a state of frenzy within three days. Inflammation of the rumen was noted.

Minot and Hall have each directed attention to the relation between the toxicity of carbon tetrachloride and the calcium content of blood. They recommend that the diet should be rich in calcium for some days before administration of the drug and that intravenous injections of

calcium chloride be given in the event of acute intoxication. In this connection it may be noted that the drug appears to be as toxic to bullocks as it is to milking cows. Carbon tetrachloride has been used against bots and worms in horses. Thompson reports that at the Remount Depôt, Fort Royal, the animals after fasting for 18 hours are given 50 c.c. and eight ounces of Glauber's salt in 800 c.c. of water, both drugs being administered by stomach tube (*Vet. Bull.*, Washington, 1933, **27,** 261). Experimental doses of 0·5 c.c. per kilo were given to horses without any ill effects (Sokola, *Jugoslav. Vet. Glasn.*, 1934, **14,** 45). On the other hand, F. Hare considers that carbon tetrachloride can be dispensed with against equine parasites, since oil of chenopodium and carbon disulphide are safer and more effective (*N. Amer. Vet.*, 1934, **15,** 37). Pelckmann has made experiments on the intoxication of horses by carbon tetrachloride with reference to the blood calcium level (*Arch. Tierhlk.*, 1930, **62,** 1). The symptoms of intoxication, which lasted ten days, were apathy, tussis, colic, diarrhœa, tremor, variations in pulse and respiration frequency and these did not depend on the amount of the drug administered. The decrease of blood calcium was independent of the dose and the symptoms of intoxication were independent of the degree of the decrease in blood calcium. "Vitakalk" given before the administration of the drug lessened the decrease of blood calcium but had no effect on the severity of intoxication. Calcium chloride prevented the decrease of blood calcium but did not prevent the symptoms. Irradiated vitamin yeast lessened the decrease of blood calcium and also the duration and severity of the symptoms. Since carbon tetrachloride produces an increase in the guanidine content of the blood, Cutler suggests that poisoning is largely due to this increase. The result is that the carbohydrate stores of the body are rapidly depleted and lactic acid increases; hypoglycæmia appears to be the immediate cause of death in dogs (Influence of Diet on Carbon Tetrachloride Poisoning in Dogs, *Journ. Pharm. and Expt. Ther.*, 1932, **45,** 209). The drug also causes a retention in the blood of bile pigments which combine with blood calcium and are excreted in this form; there is thus a deficiency of ionised calcium and consequent hæmorrhage in the serous and mucous membranes; tetanic convulsions may also be produced.

Tetrachloro-ethylene

Tetrachloroethylene, C_2Cl_4, was introduced by Hall and Shillinger in 1925 against hookworms of the dog. Its action is similar to that of carbon tetrachloride, but there appear to be no liver lesions. It is not effective against liver fluke. There is not the danger of inhalation intoxication and Hanson found it as effective as carbon tetrachloride for hookworm and ascarids in foxes. Dogs survived doses up to 15 c.c. per kilo; the margin of safety thus appears to be high. Schlingman made critical tests on puppies and found it to be safer than carbon tetrachloride. Lamson, Robbins and Ward have made an important study of the pharmacology and toxicology of tetrachloroethylene; they found no pathological changes in the liver and contrast this with the widespread destruction of liver tissue caused by carbon tetrachloride. These authors draw the conclusion that tetrachloroethylene can be used in the treatment of hookworm disease with far greater safety than either oil of chenopodium or carbon tetrachloride (*Amer. J. Hyg.*, 1929, **9,** 430). Daubney gave 0·1 to 0·2 c.c. per lb. bodyweight to sheep with no ill effects. The drug has now been used for some years, but the following are the only cases I know in which it has been suspected of causing poisoning. Twenty pigs in good condition about three months old were each dosed with 1 c.c. of tetrachloroethylene. In five hours two were dead and the next morning nine were dead. *Post-mortem* examination revealed extreme reddening of the mucous membranes of the bowels. A dog died of acute gastritis the same day as a capsule of the drug was administered. No common poison was detected in the organs on chemical analysis. Miss M. Stewart has recorded illness in a dog three hours after being given 5 c.c. of the drug, but recovery occurred in 20 hours (*Vet. Rec.*, 1934, **14,** 378).

Tetrachloroethane (*Westron*), $C_2H_2Cl_4$, is a very poisonous compound employed as a non-inflammable solvent for acetyl cellulose, fats and rubber. Daubney gave 3·3 c.c. to a sheep (33 lb.); death supervened from respiratory paralysis seven hours after administration. The same compound has caused fatalities amongst industrial workers, the liver being affected. It has been used as a vermicidal dressing, but its toxic properties are such that it is not desirable to employ it for this purpose.

Trichloroethylene (*Westrosol*) C_2HCl_3, is used for extracting oil from seeds and for dry cleaning; the vapour is less toxic than that of Westron. The published observations on the effects of this compound on sheep and cattle are in striking contrast with those of carbon tetrachloride on these animals. Daubney gave 4 c.c. by stomach tube to a sheep (24 lb.); respiration ceased in 4½ minutes, the heart beating for 14 minutes longer. A sheep (20 lb.) died 50 minutes after being given 3·2 c.c. These results may be contrasted with those of Stockman who gave three ounces of the compound to a heifer daily for 39 days without ill effects. In connection with the possible application of

this liquid as a drug for cattle and also with the cause of the "Dürener Sickness" (*infra*) it is desirable that more experiments be made with the pure drug on sheep and cattle. Trichloroethylene has been found efficient as a vermicide (*Pharm. Journ.*, 1916, 384; 1920, 243). *Dichlorobutane*, $C_4H_8Cl_2$, and *chlorobutylene*, C_4H_7Cl, have been used as anthelmintics for the horse.

Oil of Chenopodium

Oil of chenopodium is a drug possessing entirely different chemical characters from the preceding compounds, but since it is a common anthelmintic, some reference may be made to its toxicity. A sheep (40 lb.) after receiving 1·5 c.c. intravenously was thrown into convulsions and died in 23 minutes (Daubney). Sheather found that 4 c.c. by the mouth was toxic to lambs. Numerous cases of poisoning in pigs by this drug are quoted by Vajda, the symptoms being convulsions and muscular spasms (*Wien. tierärztl. Mschr.*, 1935, **22**, 142). Similar losses in pigs have been reported from America. Major Pryer found that 10 c.c. of the oil administered intravenously rapidly caused acute symptoms in a mare, but that 5 c.c. produced no constitutional disturbance in a horse (900 lb.). There have been several cases in which dogs have had convulsions and died after administration of the drug; inasmuch as no common poison was detected in the organs and no other cause of death was discovered on *post-mortem* examination, it is highly probable that the deaths were due to poisoning by the drug. After a series of fatalities from the use of the oil reported in the medical press the sale was prohibited in Germany except on a physician's prescription. The active principle of oil of chenopodium is ascaridole, the amount of which varies in different specimens.

FOODS

Whilst some foods may in themselves be deleterious when eaten in large quantities, it is more common to find injury produced by the presence in foods of such poisonous ingredients as castor seed, corn cockle or mineral poisons; sometimes the ingestion of mouldy fodder or of feeding stuff attacked by fungi has caused damage to animals. Burgi is of opinion that many of the fatalities amongst horses encountered in the cliniques of the Swiss veterinary schools were due to the ingestion of inferior or deteriorated fodder (*Tierärztl. Rdsch.*, 1932, **37**, 551).

Lathyrus sativus

Since 1925 doubt has been expressed by some writers concerning the toxicity of *Lathyrus sativus*. These doubts should be dispelled by Steyn's investigation; he found that *Lathyrus sativus* free from weeds grown at Onderstepoort and fed without any additional ration was poisonous to horses (*Onderstepoort Journ.*, 1933, **1**, 163).

Cyanogenetic Glucosides

In Great Britain poisoning by hydrocyanic acid liberated from a cyanogenetic glucoside by an accompanying enzyme is occasionally produced by animals eating the leaves of the common shrub, *cherry laurel*, but more frequently by animals being fed on foreign *linseed cake* yielding a relatively high amount of hydrocyanic acid. This trouble is usually experienced when calves are fed on such cake which has been steeped in warm water, but there have also been cases of the poisoning of horses and in France of sheep due to feeding with linseed cake containing a high proportion of hydrocyanic acid. In a paper by J. F. Tocher are described some feeding experiments made at the Rowett Institute; some of the results form a valuable contribution to our knowledge of the effects of castor seed, linseed cake and common salt on animals (*Vet. Rec.*, 1935, **15**, 477). The value of the paper would have been much greater if some reference had been made to investigations which have been carried out previously by others, but the absence of any such refefences makes it appear that the author is unaware of the many examples in veterinary literature of poisoning by the materials in question. In connection with linseed Dr. Tocher states that "this change of poisonous acid into another substance is what occurs when linseed is mixed with water. The glucoside decomposes and hydrocyanic acid is formed. It is converted very rapidly into formic acid which volatilises. The linseed therefore contains no poison whatever. This process of mixing linseed with water is well known as the method of ensuring no ill effects from the glucoside in linseed." These statements are in direct opposition to what I said at a meeting of the Lincolnshire Division in 1928: "the best temperature for the rapid generation of hydrocyanic acid is about 40°C., at which temperature about 50 per cent. of the acid is liberated in less than half an hour, the maximum amount being formed in from three to five hours. But if the enzyme is destroyed by boiling water, no appreciable amount of hydrocyanic acid is subsequently produced. It is thus clear that the conditions for obtaining the maximum quantity of prussic acid, and therefore the worst possible way of preparing the food, is to make a gruel of the meal with water and that the best way is to pour boiling water on to the meal or to add the meal to actively boiling water and allow to cool" (*Vet. Rec.*, 1928, **8**, 517). That the prussic acid generated from linseed is not rapidly transformed into formic acid is demonstrated by the results of the following experiments. Water

at 40°C. was added to $\frac{1}{10}$th of its weight of linseed cake meal; the mixture was allowed to cool to 15°C. and stand for 24 hours. The amount of hydrocyanic acid produced was equivalent to 0·058 per cent. of the meal. After standing for 48 hours the amount present was 0·067 per cent. and at the end of 54 hours it was 0·07 per cent. It is thus seen that under these conditions the quantity of hydrocyanic acid generated did not disappear in three days, but actually increased during that period. Intoxication by cyanide is usually very acute; if death does not occur within an hour or two, the animal usually recovers, but the following cases appear to indicate that a less acute form of cyanide poisoning with a fatal termination may occur. A calf was ill for about five days prior to death, the symptoms being accelerated respiration with occasional cough and dullness. *Post-mortem* examination revealed congestion of internal organs and some gastro-enteritis. Another calf died after showing similar symptoms for two days. These calves had been fed on linseed cake soaked in water for 24 hours; the cake yielded 0·05 per cent. hydrocyanic acid. Campbell reports that three mares were fed on a hot linseed mash (poulticing linseed); they showed laboured breathing and foaming and died in 15 hours, 22 hours and three days after ingestion of the linseed. *Post-mortem* examinations of two revealed that the lungs were black and gorged with blood and the stomachs were inflamed.

Clawson, Bunyea and Couch have found that the minimum lethal dose of hydrocyanic acid for sheep is one milligram per lb. of body-weight and for cattle slightly less than one milligram per lb. (*Dept. Agric. Leaflet No.* 88, Washington). Owing to the increasing frequency of cyanide poisoning in cattle in America by a number of species of wild and cultivated plants, much attention has been directed recently to the question of treatment. Hydrocyanic acid inhibits the action of the oxygen-activating enzymes; the tissues are thus unable to utilise the oxygen in their normal metabolic processes. Oxyhæmoglobin accumulates in the blood, the venous blood being bright red; death is due to asphyxiation. The action of antidotes is either to convert the cyanide into a harmless compound or to convert oxyhæmoglobin into methæmoglobin. Whilst the rapidity of action of cyanide on animals usually renders treatment of no avail, it may be mentioned that glucose in some cases has been said to give good results and that amyl nitrite, methylene blue, colloidal sulphur, sodium nitrite and sodium thiosulphate have been successfully employed. The recorded observations on the effect of glucose are contradictory. Glucose and fructose in solution combine with prussic acid under certain conditions and the resulting compounds yield hydroxy-acids and ammonia. The different results which have been obtained with glucose may be due to the fact that glucose only combines readily if the solution is alkaline; thus cyanide rapidly disappears if glucose is added to potassium cyanide solution. It has been said that an attempt was made to poison Rasputin in Russia in 1916 by potassium cyanide which had been added to port wine several hours before it was given to the monk; the poison had no effect. De Saint Rat found that after adding 5 grams of potassium cyanide to 250 c.c. of port wine, 70 per cent. of the cyanide disappeared in two hours and all of it in 24 hours. The hydrocyanic acid had thus combined with the sugars present in the wine (*Semana médicale*, 1927, **1**, 92). Experiments made at the Chemical Warfare Research Station at Porton indicated that glucose was of little value in cases of cyanide poisoning. Intravenous injections of sodium nitrite or sodium thiosulphate are effective remedies. In practice 40 to 50 c.c. of a 20 per cent. solution of sodium thiosulphate may be given intravenously to horses or cattle, the dose being repeated after a few minutes (Stainton, *Vet. Med.*, 1934, **29**, 437; Clawson, Bunyea and Couch, *J. Washington Acad. Sci.*, 1934, **24**, 369, 528).

Acorn

Acorns.—Literature contains many references to the oak, on account of the use made of the acorns for feeding pigs. The Romans used acorns for this purpose. Evelyn asserted that a peck of acorns a day with a small amount of bran would make a pig increase a pound in weight per day for two months. A few centuries ago the right of feeding swine in woods was one of the most valuable kinds of property. Fifty years ago acorn pickers were able to obtain about a shilling a bushel for the acorns, which were fed in varying proportions with bran to sheep, horses and cattle as well as to pigs. It thus appears that acorns constitute a good food for pigs. But in this country acorn poisoning in cattle has been repeatedly observed, the fatalities occurring when acorns are abundant and herbage scarce, that is to say, when acorns are almost the only food the animals receive. Feeding experiments have reproduced the usual symptoms of the disease in a steer but produced no symptoms of illness in a sheep. Few references to acorn poison are found in foreign literature, but Södermark has recently encountered 20 cases, of which 15 were fatal, in cattle in Sweden, where the disease is considered to be rare (*Svensk. Veter. Tids.*, 1934, **39**, No. 15). The symptoms are similar in oak-leaf poisoning, which occurs frequently in the United States and other countries. Gould and Morgan

have carefully observed the progress of acorn disease in mature milking cows in the New Forest area during 1933; they state that the organs had a leathery appearance and consider that the most satisfactory treatment is to give copious draughts of liquid paraffin (one half to one gallon) (*Vet. Rec.*, 1934, **14**, 33). Leonard Jones has stated that he attended 140 cattle affected with the disease in Mid Wales in 1933; most of these were young cattle and there were 80 fatal cases. Jones obtained the best results by using large doses of liquid paraffin followed by chlorodyne. The amount of tannin in acorns is 3 to 4 per cent., but some determinations in which amounts of 7 and 9 per cent. were found have been recorded; oak leaves contain 6 to 11 per cent. of tannin. The assumption has therefore been made that the damage is mainly due to tannin; this hypothesis is supported by the leathery appearance of organs noted by some observers. Marsh, Clawson and Marsh administered 25 grams of tannin daily to a bovine for 28 days without ill effect, but another which received 50 grams daily was found dead on the sixteenth day. *Post-mortem* examination showed hæmorrhages in the trachea and duodenum and hyperæmia of the lungs. The authors, however, observed no symptoms similar to those of oak-leaf or acorn poisoning in the course of their experiments (*U.S. Dept. Agric., Professional Bulletin*, 1919, No. 767).

Horse Chestnut

The composition of *horse chestnuts* (*Æsculus hippocastanum*) is similar to that of acorns, but the amount of tannin is lower (2 to 3 per cent.). The botanical name is said to have been given on account of horse chestnuts having been used as a remedy for glanders, chest complaints and broken wind in horses. There are references to their use as food in some countries, especially for fattening sheep and as a mash for poultry. I cannot say whether or no they are used at present as food for animals in England, but their composition and low tannin content indicate that they would be of value as food with less risk of damage to animals than is the case with acorns.

Soya Bean

No cases of illness due to *soya bean* have been heard of since the Staffordshire case in 1926, doubtless owing to the discontinuance of trichloroethylene for extracting the oil from the beans. Nevertheless, the complete elucidation of the disease first investigated by Sir Stewart Stockman and subsequently termed the "Dürener Sickness" in Germany might well throw light on other obscure ailments of cattle which may be due to feeding. Stockman's experiment with trichloroethylene on a heifer appeared to show conclusively that the trouble was not due to poisoning by residual solvent in the meal. But Daubney's results with the same compound on sheep show that it is desirable to extend the experiments in order that a definite conclusion may be drawn with regard to the toxicity of the solvent in question. Samin has fed to each of a number of cows during 133 days soya bean meal (1,350 lb.) which had been extracted with carbon tetrachloride; no harmful effects were produced. When soya bean meal which had been extracted with trichloroethylene was fed to cows in quantities of 5 to 16 lb. daily for four to eight weeks, it induced leucopenia with lymphocytosis (*Inaug. Dissert., Tierärztl. Hochschule*, Berlin, 1931). In his investigation Stockman considered the possibility of the disease being due to a deficiency of vital constituents in the food, but he expressed the opinion that this possibility was discredited on account of the field and experimental animals having received other foods such as hay, bran, oats or grass. The constant feeding of foods deficient in lipoids has been stated by Jaffe to lead to fatal results.

The conclusion drawn by van Kampen is that the disorders caused by soya bean extracted with trichloroethylene are not the result of residual solvent used in extraction but that they are due to the removal of lipoids from the material to an extent which constitutes a deficiency for normal milk production. The decrease in white corpuscles observed by Stang is considered to be a direct result of this deficiency (*Landw. Versuchs-Stat.*, 1929, **108**, 287). Stockman was impressed by the similarity of the disease to that suspected of being produced by the eating of bracken; profiting by the experience gained in the soya bean experiments he fed a calf with 260 lb. of bracken during 29 days and succeeded in reproducing the symptoms of suspected bracken poisoning. The question therefore again arises: is the disease caused by bracken an intoxication due to a toxic constituent or is it a deficiency disease? Outbreaks of this disease still occur; there were numerous cases this year in the west of Scotland.

Bracken

"Scabbed" Barley

A few years ago there were cases of illness in pigs in this country in which the chief symptom was vomition. It was found that these pigs had been fed on *Federal Barley* imported from America. Similar damage was noted in Germany in pigs which had been fed on American barley and several investigations were made in that country to ascertain the cause of the trouble. Feeding experiments demonstrated that the barley could be fed to horses, cattle and sheep with no ill effects, but that it rapidly produced vomition in pigs. Stang found that it was infected with *Gibberalla saubinetii*. During 1928 pigs and poultry were extensively

poisoned in Iowa by the consumption of barley contaminated with *Gibberalla saubinetii*; Roche, Bokstedt and Dickson found that the scabby barley may be fed to cattle and sheep without ill effect but that pigs, horses and dogs cannot tolerate even low percentages of badly scabbed kernels.

Salt

The presence of *salt* in excessive amounts in food is one of the most frequent causes of poisoning in poultry. Salt poisoning was the cause of the deaths of 15 silver foxes, which, after receiving heavily salted food, showed symptoms of vomition, salivation and convulsions. Each fox had ingested about two ounces of salt (Schmid, *Tierärztl. Rundsch.*, 1935, **41**, 162).

Sand

Excessive amounts of *sand* in food have been suspected of causing damage to pigs.

Powdered Glass

There is a belief in S. Africa that *powdered glass* in food produces death in dogs. Thomas and Brown made experiments in which they gave dogs powdered or broken glass in meat; they found that the ingestion of particles of glass varying in size from fine to very coarse did not produce death in dogs (*Jl. S.A.V.M.A.*, 1933, **4**, 149).

Castor Seed

Attention has recently been directed by the Royal Agricultural Society to the question of the presence of *castor seed* in some feeding cakes of foreign origin; this was considered by an Advisory Committee appointed by the Ministry of Agriculture. Time does not permit me to discuss the aspects of castor seed poisoning in this address.

Corn Cockle

Corn cockle present in food is sometimes held to be responsible for the deaths of animals. Feeding experiments have produced varied results, doubtless owing to differences in the rates of absorption of the toxic saponins by the intestinal mucous membranes of the animals. Berner's investigation of corn cockle poisoning in the horse is a valuable contribution to this subject (*Zeitschr. für Veterinärkunde*, 1930, Jan.).

POISONOUS PLANTS

The review by Craig and Kehoe presented at the Annual Congress of the N.V.M.A. ten years ago and Dr. W. H. Andrews' opening of the discussion thereon render it unnecessary for me to deal with this source of poisoning amongst animals (*Vet. Rec.*, 1925, **5**, 795, 816). Although this is probably the most interesting branch of veterinary toxicology to country practitioners, it is for various reasons the most unsatisfactory chapter. In this connection I may quote Dr. Andrews: "There are very wide gaps in our knowledge of poisonous plants and much experimental work is essential if we are to fill them. Research on such problems is apt to be expensive, more especially since it would be necessary to utilise some of the domestic animals themselves, and not small laboratory animals, as experimental subjects. There are many points which can only be settled by carrying out properly controlled tests on suitable animals and other aspects of the problem require the attention of the chemist and botanist. The greatest success will always be attained through the co-operation of laboratory and field workers." In 1927 the Ministry of Agriculture and Fisheries regarded the question as to the extent to which plants are poisonous on the farm as so important that they commissioned one of their officers, H. C. Long, to prepare a volume on the subject. Long expressed the view that "deaths are sufficiently numerous to make it certain that financial losses are in the aggregate very heavy." The results of some investigations on certain poisonous plants have been published by the staff of the Institute of Animal Pathology, Cambridge, and further contributions from the Cambridge school will be awaited by practitioners and others interested in such research. In conclusion, I hope that members of the veterinary profession will offer their co-operation and that the Agricultural Research Council will give assistance and encouragement to workers in this field, so that some of the wide gaps in our knowledge may be filled with a consequent diminution of the financial losses to agriculturists.

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Discussion

The President (Mr. H. Fraser), in declaring the paper open for discussion, said he was convinced there were more cases of plant poisoning in animals than it was generally realised.

Mr. F. T. Harvey agreed and instanced the death of some cattle after they had eaten a quantity of leaves from beech trees. He referred to the analysis of the hair of animals to prove arsenical poisoning. It had been proved that there was arsenic in hairs removed from the tail of a beast which had not taken arsenic into its body for nine months. He believed that an analysis of bone had been tried in an attempt to prove the presence of lead in the tissues.

Mr. Esmond Brown said he was greatly interested in arsenical poisoning, but when he heard of the finding of one four-thousandth part of a grain in the hairs of an animal he wondered if that was sufficient proof. He thought there might be enough arsenic almost anywhere in the country to produce that amount, and that any animal which had been grazing in such parts was likely to have some in its hair. Referring to arsenical dips, Mr. Brown said that the incidence of deaths was not entirely dependent on the strength of the dip used. There were other factors such as the treatment of the sheep before dipping. He had, however, seen sheep which had been rough-handled and thrown

into a dip far over strength and nothing had happened. But in other cases, where it appeared that instructions had been very carefully carried out, there had been deaths. Deaths were apt to fall into groups—sometimes pure-breds were affected and not cross-breds, and *vice versa*.

There might be some importance attaching to the kind of skin one found in individuals and groups.

He had found that there were many more losses at the second dip than at the first. He thought this was probably due to a disturbance of the skin at the first dip which rendered the animal more susceptible at the second.

With regard to plant poisoning in general, laboratory experiments were not complete, as they did not produce the natural conditions. So-called bracken poisoning and a good many other plant poisonings frequently occurred in a period of drought towards the end of summer. It might be that animals were driven during an abnormal weather period to eat plants which they would not otherwise touch; also that the animals got certain plants in exceptional quantities, or that some plants were changed somewhat in composition from that of a normal season. With regard to *Lathyrus sativa*, it sometimes produced poisoning and sometimes it did not. Dr. Clough had mentioned that salt was known to have caused poisoning in poultry. It certainly also caused poisoning in swine and he had seen a number of cases in which he was convinced that death and illness among pigs were attributable to salt. Washing soda also caused trouble.

Major W. Fyrth referred to trouble in swine which he thought was due to Federal barley.

Mr. R. H. Smythe said he was particularly interested in the question of poisoning arising from industrial processes.

During the last 27 years he had practised in the mining district of Cornwall and he supposed he could claim to have had one of the largest measures of experience of arsenical poisoning in all its forms. They had to differentiate between the fumes which arose from smelting processes and those from calcining processes. He knew of one large smelting works from which an appreciable quantity of arsenic did not escape from the stacks but from which the fumes were acid and the grass in the district was burnt. He thought it probable that the calcium content of the soil was diminished by the presence of the acid. One farm just under the stack could grow only coarse grass. After the works shut down grass grew well and the quality was good. While the land was contaminated by acid and the soil was probably deficient in calcium there was never a case of any kind attributable to calcium deficiency. A case of milk fever had never been known on that farm.

There used to be several types of arsenicaf poisoning. Among the acute cases they saw were some in horses carting arsenic: one horse following the cart of another apparently licked it. In another type of case horses lost hair and wasted away, but still more common was it to find horse after horse developing staggers and dying, with no other symptoms. There was a very peculiar third type in which the symptoms were loss of hair and emaciation. At times a horse would go to a stream containing a mixture of arsenic, sulphur and iron and take a good drink. Within an hour it would be nearly asphyxiated with its tongue hanging out of its mouth, and its breathing could be heard many yards away, death from asphyxia resulting. It was a condition he had not seen recorded.

With regard to insoluble arsenical compounds, he thought this problem presented more difficulty in the mining districts than any other. Years ago all cases of illness or death that occurred in the mining area were attributed to arsenic—just as long as the mines were paying compensation.

It was very debatable whether mundic was poisonous in its insoluble form, for in some cases handsful of mundic had been taken from the stomachs of cows which had not been ill. Arsenical fumes not only contaminated the surrounding district, but also grass and hay which might be taken miles away. He had seen cases which were the result of hay from contaminated areas. In the affected areas arsenic could always be obtained by brushing the hair.

With regard to lead poisoning, Mr. Smythe mentioned three dogs which died over a period of years. It was discovered that the dogs had been drinking from a slate trough which had a lead outlet pipe. Analysis showed the water to contain a considerable quantity of lead. In another case a dog was poisoned by water from a hot-water tap fitted to a lead cistern. In this instance a boy drinking from the same supply was taken ill but recovered.

Following a rat destruction campaign by a local authority which was using phosphorus poison spread on bread, he had seen deaths of dogs, sea-gulls and jackdaws.

Mr. L. E. Perkins spoke of cases in the North of England attributed to poisoning by potatoes. A man had a large quantity of potatoes left over and he fed them to dairy cattle. He did not see the cases in the acute stages, but he saw them just after and kept in touch with them for about two months afterwards. The symptoms he noted were acute diarrhœa closely resembling that of Johne's disease, marked emaciation, weakness, loss of milk, tightness of hide and the general appearance of unthrifty animals. As soon as the acute stages appeared the owner stopped feeding the potatoes, but it was some time before the animals regained their normal condition.

He had recently seen a case in a second-calf Guernsey cow which had gone right down in condition. She had been receiving five pounds of potatoes per day, and at first sight looked like a typical case of Johne's disease. The potatoes were discontinued and the animal had improved considerably in two or three weeks. It appeared to have been a case of potato poisoning. He wondered what exactly was the active principle in potato poisoning? As far as cattle were concerned he did not know. It appeared that if they gave an over-dose of potatoes and continued to do so, distinct poisoning was caused, but small quantities given over a longer period produced no ill effects.

Mr. C. V. Watkins alluded to a case in which waste products from a cider factory near a farm ran into a stream, which resulted in the exhibition of symptoms resembling botulism in cattle.

He also mentioned poisoning in cattle suspected to be due to the ingestion of *Iris fœtidissima* seeds. The suspected outbreak occurred in the fall of the year when the seeds were very numerous, and the cattle sheltered during southerly gales under trees where the plants were growing. There was evidence of the plants having been eaten, but it was not proved that the seeds were toxic. Did Dr. Clough know of any poisonous effects of these plants?

Mr. L. W. Bovett spoke of trouble among pigs which the owner was afraid was due to feeding stuffs. The speaker thought it was some other cause. The pigs were housed in galvanised sheds and while there was no rain there was no trouble. When it rained the animals were given water which was collected from the roof of the shed. When

they stopped the water there was no more disorder. He asked if the soft water running over the galvanised surface could become harmful?

The Hon. Secretary mentioned some cases of arsenical poisoning by weed killer in which several fowls and a donkey were killed. The donkey, fowls and some ducks had access to water contaminated by the poison. There were no losses among the ducks.

He asked if it were correct that there were about 13 kinds of bracken, only one of which was poisonous.

The Reply

In reply, Dr. Clough said that he agreed with the President and Mr. Harvey in their view that there are probably many more cases of plant poisoning than are suspected. But in the past there had certainly been a number of cases in which plant poisoning was suspected, when investigation subsequently proved that the damage was due to other causes. Poisoning of horses by beech nut meal had been reported; he was not aware whether the toxic constituent of the seeds was present in the leaves. He thought that the specimen of hair submitted by Mr. Harvey some years ago was the first from an animal which had been tested with a positive result; human hair had, of course, often been tested for arsenic.

In reply to Mr. Brown, he did not think that arsenic would be detected in hair unless a relatively considerable amount had been absorbed over a period of time. In the S. African experiments it was carbolic dip which was shown to have been absorbed by the skin; it was well known that arsenic from a sheep dip was only absorbed by intact normal skin in relatively small amount. Owing to repeated failure to find excessive salt in pigs' stomach contents, he had sometimes thought that salt poisoning in pigs was rarer than was commonly supposed; but Mr. Brown's experience indicated that it was not uncommon; possibly chemical analysis was not of much value in diagnosing this form of poisoning, as most of the salt might be absorbed before death.

Mr. R. H. Smythe's experience in the Redruth mining district was especially valuable, since the judgment in questions of poisoning in such a locality must ultimately be given by the trained observant clinician. He (the speaker) suggested that the very insoluble arsenical pyrites in a coarse physical state was not absorbed when taken by an animal, but that it might be slowly oxidised by air in the presence of water, yielding a less insoluble form of arsenic. Possibly lead poisoning in dogs was not as uncommon as he had thought; he could only judge by cases submitted for analysis. Incidentally, he might mention that although many cats were sent for analysis, it was seldom that any poison was found in them; but recently mercury had been found in several.

Mr. Perkins had directed their attention to potato poisoning; the symptoms described in different cases showed that the cause was not always the same.

Mr. Motton had asked how many varieties of bracken existed; only a botanist could answer this with authority. Since bracken poisoning had been experimentally demonstrated in England, America and other countries, it seemed wise to regard all varieties with suspicion. Mr. Watkins suggested that botulism might be the cause of damage to animals by vegetable waste; this suggestion should be kept in mind, but there was undoubtedly a large amount of sulphuretted hydrogen produced from fruit under some conditions of fermentation. Compared with arsenic and lead, zinc had low toxicity and he did not think that sufficient would be dissolved by soft water flowing over galvanised iron to produce poisoning in animals. Owing to our lack of knowledge of the poisonous principles of plants, chemical analysis had at present a very restricted value in diagnosis of plant poisoning. Even when the toxic principle was known there were many difficulties. For example, hemlock contained the volatile alkaloid coniine, but there were invariably volatile bases in *post-mortem* specimens and their chemical characters were not unlike those of coniine. Physiological experiments were untrustworthy, as many other organic bases showed similar physiological action on small animals to that of coniine.

The Veterinary Record

January 18th, 1936.

VETERINARY TOXICOLOGY

THE address by Professor Clough printed in this issue again draws attention to the increasing interest displayed in the subject of veterinary toxicology. The veterinary is wider than the human aspect of this branch of science in that poisoning is more common among animals and, especially as regards plant poisoning, covers a wider range. So far, however, veterinary toxicology lacks in a great measure the sure foundation of exact pharmacological research—based, it is true, on tests on animals—which supports our knowledge of poisoning of the human subject. The two branches have much to give and take in building up the stock of common knowledge.

In recent years a great deal has been done, and is being done, both in the Dominions and at home, towards the acquisition of precise information on the active principles, physiological effects, and those most important and obscure factors governing the poisoning of stock. We trust that this excellent work will go forward and continue to secure adequate official encouragement and financial support. The progress of science is slow and it is futile to expect practical results without prolonged and intensive work. In the long run patience will be rewarded, our opinions will be better based, and valuable remedial and preventive measures will emerge, which will make the labour and expense well worth while.

In our branch of the subject it is perhaps well to reflect that the practical interest involved is usually financial, whereas in human toxicology it is moral or ethical or in grave events criminal. This inevitably leads to a less thorough and rigorous scientific investigation

into facts as opposed to allegations in regard to damage done to animals, than is essential in regard to man. Even a court of law, which exists for the purpose of eliciting truth as a preliminary to dispensing justice, is in practice liable to rely on evidence in cases of alleged damage to stock which would not be accepted save after most careful scientific study in a criminal issue. It may well happen that decisions are made on the foundation of the *bona fide* but inexact observations of practical men; on the fallacious reasoning that what is æsthetically objectionable and therefore damaging to us, is therefore also damaging to an ox, or a pig, or a sheep; or on the *ipse dixit* of an analytical chemist in general practice who does not possess specific knowledge of this particular subject. Many "scientific" witnesses, for example, adopt quite arbitrary standards, asserting that a proportion exceeding so and so is dangerous, and projecting considerations which may have relevance to man into the animal kingdom. A truer sense of proportion and a far wider knowledge of comparative physiology are very badly needed. Again, as our readers will understand, experimental and particularly field tests are not always conducted in a vigorous manner. It is sufficient to remind ourselves of the commonest fallacy—namely, the variation of more than one independent variable at a time. So-called practical tests on sewage poisoning have well illustrated this point.

Another matter which in our view is of great importance is the necessity for the prosecution of more research into the erratic incidence of many poisonings. Here carbon tetrachloride, acorn, bracken, soya, and castor seed may be cited. So far as castor seed is concerned, we fear that there may be a tendency to regard it as harmless by reason of the fact that many cases of its consumption without ill effect are observed, whereas a careful search of the literature will reveal thoroughly well-documented cases of damage. In no circumstances, therefore, would it be wise to exonerate the seed or to regard its use as other than a grave potential hazard. We shall again in time and by virtue of patient research know more of this problem, but in the meantime attention may usefully be drawn to the significant observations of Ewart on the incidence of saponin poisoning, which suggest a correlation between the condition of the alimentary tract and the absorption therefrom of indiffusible substances, or substances diffusible with difficulty, of the saponin or toxine types.

Another question which cannot be regarded as by any means settled is the possibility of chronic plumbism in stock, having regard to the average time required for its production in man and the average duration of the life of livestock.

But a mere outline of the outstanding problems would lead us far beyond the limits of a leading article, and we must rest content with welcoming the interest displayed, and the publication in our literature of practical addresses such as that of Professor Clough.

Clinical Communication

BOVINE PIROPLASMOSIS: A COMPARISON OF THE RESULTS OF TREATMENT WITH TRYPAFLAVINE AND TRYPAN BLUE

S. G. WILSON, B.Sc., M.R.C.V.S.
District Veterinary Officer
Lilongwe, Nyasaland

Disease was reported amongst a herd of European-owned, chiefly "grade," cattle of mixed age recently transferred from a farm about 200 miles distant. During the latter part of their journey they escaped the routine arsenical dip and deaths occurred eight days after arrival at the farm, or 24 to 26 days after previous dipping.

When visited on October 13th, the animals were then being dipped in an adjoining Government dipping tank, but were still tick infested, the following species being present: *Boophilus decoloratus* (numerous), *Rhipicephalus capensis* (few), *Amblyomma variegatum* (numerous) and *Amblyomma tholloni* (few). The last-named tick is new to the Protectorate.

Symptoms.—Owing to the time of year, food and water were scarce, therefore the general condition of the herd as a whole was poor. The native herd boys detected sick animals by inappetence and hollowness of the flanks, the latter being an early marked symptom. The other symptoms customarily accompanying debility and anæmia were noted. In no case were the mucous membranes jaundiced even a few hours before death. In the limited number of cases noticed in various outbreaks a jaundiced mucous membrane invariably means, on microscopic examination, a mixed infection, say anaplasm infection complicating a *T. vivax* or a piroplasm infection.

Blood Examination. — This showed pure *P. bigemina* infection, round and pear-shaped forms predominating. In one corpuscle a triangularly-shaped form accompanying a pear-shaped form was seen. It was impossible to

state the percentage of corpuscles invaded, the extent merely being noted as a light or heavy infection.

Treatment.—Five-day dipping (hand dressing in weak cases) was commenced and the animals were divided into two lots, one lot being treated with trypaflavine, the other with trypan blue. A few left as controls died.

A.—Trypaflavine.

Case.	Subject.	Date.	Temperature.	Respiration.	Pulse.	Condition.	Mucous Membranes.	Blood Examination.	Treatment.	Result.	Remarks.
1	Heifer	14.9.35	107·5° F.	17 and laboured	92	Serious	—	Most erythrocytes invaded	60 c.c. trypaflavine intravenously	Died	Noticed ill the previous morning. Not able to rise after injection. Died in a few hours.
2	Heifer	,,	104° F.	Very fast	64	Not so serious	Not so pale as No. 1	Light invasion	60 c.c. trypaflavine intravenously	Survived	Noticed ill on previous evening.
3	Friesland Cow	,,	107° F.	Fast	—	Fair	—	Heavy invasion	60 c.c. trypaflavine intravenously	Survived	Noticed ill on visit.
4	One case	,,	Normal	—	—	Better. Struggling when cast. Inappetence and hollow flanks	Good	Light invasion	60 c.c. trypaflavine	Survived	Noticed ill on visit. Showed after effects in œdematous swelling of dewlap and jaws.
5	Black Heifer	16.9.35	104·4° F.	Hurried	—	Serious during injection	Pale	Medium invasion	As condition serious during injection only 30 c.c. trypaflavine given	Died	Noticed ill on previous day. Respirations became distressed and death followed in a few hours.
6	Black cow	16.9.35	—	—	—	Not so serious as No. 5	—	Medium invasion	60 c.c. trypaflavine	Survived	Noticed ill on visit.
7	Ox	21.9.35	104·2° F.	—	—	Prolapse of penis. Serious	—	—	60 c.c. trypaflavine	Died	Ill the previous Thursday evening. Died in about 1½ hours.
8	Black ox	,,	105·2° F.	—	—	Serious	—	—	60 c.c. trypaflavine	Died	Ill the previous evening. Died in 24 hours.
9	Bull	,,	107° F.	—	—	Fair	—	—	60 c.c. trypaflavine on 21.9.35	Died	Noticed ill on visit. Seen on 24th. Weak but temperature normal. Drinking and eating morning and evening.*
10	Young Friesland	,,	106·2° F.	—	—	—	—	—	60 c.c. trypaflavine	Survived	Noticed ill on visit. Seen 3 days later: eating but still weak.

*On 27.9.35 the death of the bull was reported, following a history of general weakness with blood in fæces. On *post-mortem* examination the peritoneum was jaundiced (not to any marked extent) and fluid was present. The abomasum showed ulceration with blacked raised edges, each ulcer being about the size of a half-crown. Ulceration was less marked on the intestines but common on the cæcal lining. These ulcers did not show black edges. Both kidneys showed a mottled appearance due to uniform pin-head petechiæ over the entire surface. The spleen was normal. *Fasciola gigantica* were numerous in the bile-ducts.

B.—Trypan Blue.

Case.	Subject.	Date.	Temperature.	Respiration.	Pulse.	Condition.	Mucous Membranes.	Blood Examination.	Treatment.	Result.	Remarks.
1	Red heifer	14.9.35	107·5° F.	—	—	Fair	Fair	—	90 c.c. 2% solution trypan blue intravenously	Survived	
2	Red heifer	,,	—	—	—	Good	—	Positive	120 c.c. trypan blue intravenously	Survived	
3	Black cow	,,	—	—	—	Good	—	Positive	120 c.c. trypan blue intravenously	Died	Died in a few hours due to collapse.
4	Roan heifer	24.9.35	105·5° F.	—	—	Serious	—	—	100 c.c. trypan blue intravenously. 30 c.c. trypan blue subcutaneously	Survived	Unable to rise after injection.
5	Heifer		—	—	—	Good	—	—	Same as above	Died	
6	Cow in calf		—	—	—	—	—	—	100 c.c. intravenously	Survived	
7	2 cases	28.9.35	—	—	—	—	—	—	100c.c. 1½% solution trypan blue intravenously	Good recovery	Treated by native.

Conclusions

With trypaflavine, Cases 1 and 7 were in a hopeless condition and do not give a fair indication of the results of this method of treatment.

In Case No. 5 the end was certainly accelerated, but this may have been a case of idiosyncrasy.

In Case No 9 the lesions seemed to point to poisoning. Mash's test did not show arsenic. In this connection, it would be interesting to know the fate of trypaflavine in the body and its mode of excretion.

In recovered cases, native herd boys invariably reported the beast to be eating on the second morning or evening and thereafter progress was uneventful, except in Case 9, although in a few cases blood in fæces was recorded, but this soon passed off. In general, the young stock were attacked first, which seemed to point to a previous infection. Also, it is more than probable that the locality where the infected ticks were picked up abounded in native cattle. These must either have shown an immunity, or infection in the herd may have been, *inter alia*, due to carriers and the increase in tick fauna due to non-dipping. The former seems the more likely postulate. The idea of a breakdown in immunity tolerance may be disregarded. Trypaflavine seems to aggravate serious conditions. (Compare Guerrere and Patine, "Bovine Piroplasmosis of Colombia," extract *Vet. Bull.*, Vol. 4, page 506.) If given in the early fevered state (106°F. to 108°F.), when disease is possibly of not more than 24 hours' duration, the results are hopeful. In this connection it should be pointed out that detection of sick animals depended chiefly on native herd boys, therefore the exact duration of the disease before injection was often vague. Of the entire herd, only one-third became affected. The only advantage the use of trypaflavine appears to possess over that of trypan blue is the smaller dose of the former needed. Five-day dipping succeeded in eradicating the ticks except *A. variegatum*, which proved very resistant and still persists.

I am grateful to Captain Culhane, Acting Chief Veterinary Officer, for his help and advice.

Reference.—Bedford, G. A. H. Report of the Director of Veterinary Services, South Africa, 1932.

A horse-flesh restaurant and snack bar is the latest amenity at Prague. All the dishes and snacks will be of horse-flesh, which is not cheaper than other kinds of meat, but is, it is claimed, tastier and more tender than beef. The new restaurant is equipped in the most elegant modern style, and is expected to attract many patrons. Prague already has a goose restaurant where 40 different dishes all made of goose are served.—*Reuter.*

Abstracts

[Treatments for Cyanide Poisoning of Sheep and Cattle. Bunyea, H. (1935.) *J. Amer. Vet. Med. Ass.* **86.** 656-661.]

A study has been made of the most suitable antidotes to be used in cases of cyanide poisoning in cattle and sheep. Varying amounts of potassium cyanide were administered to these animals and the lethal dose, calculated as hydrocyanic acid, was determined. It was found to be 2·042 mg. per kg. body weight for cattle and 2·315 mg. per kg. body weight for sheep. Symptoms of poisoning appear in a few minutes after the administration of a fatal dose by the mouth. There is an increase in the rate and depth of respiration and quickening of the pulse, spasmodic muscular movements, respiratory convulsions, dyspnœa and death. On *post-mortem* examination no constant lesions were found, the more common lesions being congestion or hæmorrhage in the lungs. The most effective remedies were found to be the intraperitoneal injection of sodium thiosulphate and sodium nitrite; the doses being, for sheep, 1 g. of sodium nitrite and 2 to 4 g. of sodium thiosulphate, and for cattle 2 to 3 g. and 10 to 20 g. of these drugs respectively in 10 per cent. aqueous solutions. The injection of sodium thiosulphate may be repeated if necessary. To be successful treatment must be given within four minutes after the onset of symptoms; even so, there is little prospect of success if more than two minimal lethal doses have been taken.

J. A. N.

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[Additional Observations on the Toxicity of Crotalaria spectabilis (Roth) for Swine. Emmel, M. W., Sanders, D. A., and Henry, W. W. (1935.) *J. Amer. Vet. Med. Ass.* **87.** 2. 175-176.]

The authors record three cases of poisoning of pigs, kept under field conditions, by *C. spectabilis* which is grown in the southern states of America as a cover crop on poor soils. It appears that pigs are more likely to be poisoned by the green foliage of the plant than by the seeds and these animals will eat the leaves of the plant readily. As the plant withstands heavy frost, heat and drought much better than other fodder crops, the most dangerous period is following such climatic conditions.

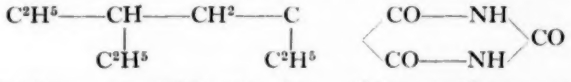
J. A. N.

University of London.—The list of Staff Examiners and Moderators, other than Medical, for 1936, published in the current *London University Gazette*, includes the names of Professor T. G. Browne, M.R.C.V.S. (Veterinary Anatomy), Colonel A. G. Todd, C.B.E., D.S.O., M.R.C.V.S. (Veterinary Hygiene) and Dr. J. T. Edwards, D.Sc., M.R.C.V.S. (Veterinary Pathology).

[A Contribution to the Study in the Dog of the Toxic and Narcotic Actions of a Derivative of Malonylurea, Etho-2-Butyl-Ethyl Barbituric Acid. (Trans. title.) Panisset, L., and Grasset, E. (1935.) *Bull. Acad. vét. France.* **8.** 2. 105-118. 2 tables. (12 refs.)]

The authors refer to the studies made by other workers of the value of the barbiturates as anæsthetics in small-animal surgery. The work of Auchterlonie with evipan-sodium and that of Wright and Oyler with nembutal receive special mention.

The authors describe their experiences with a product made in the Pasteur Institute by Fourneau and Matti. The product etho-2-butyl-ethyl-barbituric acid has been given the short title of etho-butyl and is a derivative of ethyl-butylic alcohol. It has the structural formula,



Various experiments by other authors are quoted in which the drug was used on rats, mice and rabbits. Based on the results of these experiments an endeavour was made to employ the drug in canine patients. The drug was administered subcutaneously, intraperitoneally and intravenously, but details of the results obtained by its subcutaneous administration are not given. The drug was always administered in a 1 per cent. solution.

Reference is made to the necessity of making the intravenous injection slowly and in this connection the work of Auchterlonie and that of Wright and Oyler are again quoted.

Extensive details of the use of the drug in 11 cases by the intraperitoneal route and 13 cases by the intravenous route are given in Table I. The results are summarised in the second table. Unfortunately there appears a most deplorable error in the dosage quoted in the second table, which, if the text of the article and the first detailed table are correct, should read "dose in grammes per kilogramme" and not "dose in centigrammes per kilogramme."

The results obtained when the drug was administered intraperitoneally were not satisfactory. The doses varied from 0·015 gramme to 0·10 gramme per kilogramme of body weight; with the latter dose the dog died in one hour and fifteen minutes. With the smaller intraperitoneal doses a variable degree of narcosis was produced.

Intravenously the dose varied from 0·02 gramme to 0·08 gramme per kilogramme of body weight. The two animals which received 0·08 gramme per kilogramme of body weight died immediately, as also did that one which received 0·06 gramme per kilogramme.

All the animals receiving a dose of 0·05 gramme or less per kilogramme survived.

Though the authors admit that the number of cases is not sufficient on which to base a definite opinion, they suggest that the dose of this product should be from 0·03 to 0·04 gramme per kilogramme of body weight. But with these doses, while a deep degree of narcosis is produced without any preliminary excitement, the duration of narcosis is very variable; it may last 40 minutes, but in other cases it may last four or five hours. In some of the cases a state of anæsthesia was produced. The details of the cases substantiate the statement that the duration of narcosis depends directly on the amount injected, but the depth of narcosis does not appear to bear such a direct relationship to the size of the dose in relationship to body weight.

It would appear that, as with other barbiturates, the only safe method of using the drug, as a general anæsthetic intravenously, is to inject the solution *very* slowly and to stop the injection as soon as the desired degree of anæsthesia has been produced.

[The drug does not appear to possess the merits of evipan-sodium or nembutal.]

G. F. B.

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[General Anæsthesia of the Horse with Ethyl-butyl-ethylbarbiton. (Trans. title.) Marcenac, Carnus and Comby. (1935.) *Bull. Acad. vét. France.* **8.** 2. 149-152. (7 refs.)]

One of the authors of this report published in 1930 an article on the use of chloral hydrate as a general anæsthetic for the horse. Though expressing complete satisfaction with chloral hydrate after using it in more than 500 cases, the authors decided to investigate the possibility of using one or other of the barbiturates.

The prolonged period of narcosis produced by the majority of the barbiturates renders them unsuitable for equine anæsthesia. The authors investigated the possibility of using the product etho-2-butyl-ethyl-barbituric acid, prepared in the Pasteur Institute, the action of this drug on dogs being described by Panisset and Grasset (*Ibid*).

The drug was given to horses intravenously in a 10 per cent. solution. It is stated that in this concentration the drug produces no untoward effects either on the cells of the blood stream or on the endothelial lining of the vessel, nor does it produce any marked vaso-dilatation.

The importance of making the injection slowly is stressed, otherwise there is a risk of bulbar shock.

After some experimentation, they found that 0·01 or 0·015 gramme per kilogramme of body weight were insufficient doses but that 0·02 gramme per kilogramme of body weight was a satisfactory dose. They state that with the latter dose the results were excellent; profound narcosis ensued immediately and it was possible to conduct any surgical operation during a time varying from 50 to 70 minutes after the drug was administered. It is not stated that the animal was actually in a state of deep surgical anæsthesia.

The animal recovers consciousness and is able to rise in from 90 to 105 minutes. These statements are based on observations made on 16 cases.

G. F. B.

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[Premunition of Cattle against Johne's Disease. Vallée, H., Rinjard, P., and Vallée, M. *Rev. gén. Méd. vét.* **43.** 516. 777-779.]

In 1926, Vallée and Rinjard published a preliminary note, putting forward the view that subcutaneous inoculation of living unattenuated Johne's bacilli (5 to 10 mgm. of culture), suspended in a mixture of vaseline and powdered limestone, whilst innocuous, gave protection against natural infection. An inflammatory reaction appeared at the site of inoculation, but gradually became hard and insensitive. So long as this nodule persisted, the animal appeared to be immune.

Vaccination results collected from the field are now given from 1925 to 1934, the number vaccinated annually rising from 803 to over 12,000. Altogether, over 35,000 animals have been vaccinated, with no untoward sequel. The duration of immunity is believed to vary directly with the persistence of the focus of infection, which is usually from eight months to two years, although persistence for six years was noted in one case. On 133 very heavily infected farms, with a total of 5,650 head, no case of Johne's disease was reported. The only failures recorded were in animals already infected, in which the reactionary focus disappeared in two or three months by resorption or suppuration.

The authors conclude that the vaccine is safe and effective, but recommend the prompt slaughter of all clinically affected animals, which are the source of infection.

A. W. T.

FLOOD LOSSES AT LYDNEY

In the extensive flooding caused by the rivers Severn and Wye bursting their banks during the recent great gale, hundreds of sheep were drowned at Lydney, Gloucestershire, their carcases being piled up on the railway line. Horses were marooned on the top of hayricks, and a colt was born on the top of one stack.

The sudden rush of water swept the animals away and drowned them in batches, though the farmers and their men made desperate efforts to save them. One farmer alone lost nearly 50 sheep.

N.V.M.A. Divisional Report

WESTERN COUNTIES V.M.A.*

MEETING AT TAUNTON

A meeting of the Western Counties Division of the N.V.M.A. was held at the Great Western Hotel, Taunton, on October 24th, 1935. The President, Mr. H. Fraser, occupied the Chair, and there were also present: Messrs. R. B. Nelder, Richardson, Perkins, G. A. Roberts, R. H. Smythe, F. T. Harvey, Blackwell, L. W. Bovett, E. R. Edwards, Macdonald, Menzies, Watkins, Masson, A. G. Saunders, Hall, Chase, Thorburn, Irvine, Baird, Esmond Brown, H. K. Roberts, E. W. Ascoli, C. R. Parsons, T. Shaw, A. Mather, E. S. Martin, W. Tweed, S. J. Motton, Majors Ascott, James and Fyrth, and Dr. Clough.

Apologies for absence were received from Sir F. Hobday, Major Dunkin, Messrs. Bloye, Wilkins, Gibbings, A. R. Smythe, J. Forbes, Willing, Crowhurst, Barbour, Patterson, Kauntze, Stephens, G. Harvey, C. Head and Colonel Head.

The minutes of the March meeting at Exeter, having been published in *The Veterinary Record*, were taken as read after a correction had been made (the name of Mr. E. R. Edwards as Hon. Auditor had been omitted). The minutes of the June meeting were read and signed as correct.

Obituary.—Before commencing the general business of the meeting, the President referred to the sad loss sustained by the Division by the death of one of its oldest members, Mr. W. B. Nelder. A leter of sympathy had been sent to Mrs. and Mr. Nelder, and as a mark of respect he asked those present to stand in silence for a minute.

Resignation.—The resignation of Mr. J. G. Crowhurst, who had left the district, was received with regret.

New Members.—On the proposition of Mr. Thorburn, seconded by Major Fyrth, the following gentlemen were unanimously elected members of the Division: Messrs. Chase, Mather, Wm. Tweed and T. Shaw.

Correspondence.—A letter from the N.V.M.A. *re* the payment of the expenses of the representatives of Divisions was read and discussed.

The Hon. Secretary was requested to discuss the matter with the Council of the Division and to report at the next meeting.

Paper.—A comprehensive and very instructive paper was read by Dr. Clough, D.Sc., of the Royal Veterinary College, London, on "The Sources of Some Poisons and Their Effects on Animals," this being heard with much appreciation and well discussed. [Dr. Clough's paper is reproduced, together with a report of the discussion and the essayist's reply, at the commencement of this issue. The subject under discussion is also dealt with in our editorial columns.—*Editor.*]

Votes of Thanks.—All of the speakers had expressed their high appreciation of Dr. Clough's kindness in coming to the meeting and bringing a paper of such great practical value, and before the meeting terminated, the President re-echoed the members' sentiments of gratitude to Dr. Clough.

The meeting closed with an expression of thanks to the President for conducting the meeting.

S. J. Motton, *Hon. Secretary.*

* Received for publication December 27th, 1935.

Notes and News

The Editor will be glad to receive items of professional interest for inclusion in these columns.

Diary of Events

Jan. 22nd.—Meeting of the Derbyshire Division, N.V.M.A., at Sutton Bonington, Loughborough, 2.30 p.m.

Jan. 22nd.—Meeting of the Section of Comparative Medicine of the Royal Society of Medicine at the Society's House, 1, Wimpole Street, W., 5 p.m.

Jan. 25th.—Meeting of the Scottish Branch, N.V.M.A., at Perth, 2 p.m.

Jan. 31st.—Annual General Meeting of the Yorkshire Division, N.V.M.A., at the Hotel Metropole, Leeds, 3 p.m.

Jan. 31st.—Annual General Meeting of the Royal Counties Division, N.V.M.A., at Reading, 5 p.m.

Mar. 13th.—Annual Dinner and Dance of the Yorkshire Division, N.V.M.A., at the Guildford Hotel, Leeds.

Sept. 14th-18th.—N.V.M.A. Annual Congress, Scarborough.

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PERSONAL

University of Capetown Honours
Sir Arnold Theiler

The most impressive ceremony of Graduation Day at the University of Capetown last month was the conferring of honorary degrees on five men who have done much to further education, art and science in South Africa, amongst whom was Sir Arnold Theiler, K.C.M.G., who received the honorary degree of Doctor of Science. Sir Arnold Theiler's record of service in South Africa dates back to the days of the South African Republic. On his retirement in 1927, he could look back on an unbroken record of 36 years' work as head of the Department of Veterinary Research and Education, first in the Transvaal and then for the whole of the Union.

A marble plaque has recently been affixed to the birthplace at Villeneuve-le-Guyard of Professor Chauveau, who was born there in 1827 and died in Paris in 1917. At the time of his death, he was a member of the Academy of Medicine, honorary professor of the Veterinary School at Lyons, professor at the Paris Museum,

inspector of the French Veterinary Schools and Grand Officer of the Legion of Honour.

Children of Waterloo Veterans.—Mr. Robert J. Clifford, of Merstham, Surrey, contributing to the current correspondence on this subject in *The Times*, writes: "My wife, now in her 77th year, is the only surviving child of the late Thomas Salusbury Price, M.R.C.V.S., of Kennington, who was born at Pentre Voelas, Caernarvon, in 1785, and was gazetted lieutenant in the Regular Militia of the county of Caernarvon on October 6th, 1810, his commission being signed by Viscount Bulkeley, Lord Lieutenant of the county. He served with his regiment at the Battle of Waterloo when just about 30 years of age. His commission and the sword he carried on that historic occasion are his daughter's most treasured possessions. He died on October 6, 1870, at the age of 85, on the 60th anniversary of his appointment, and my wife is the last child of his third wife, being born in 1859."

Marriage.—Mackeown—Hayes: On January 10th, 1936, at All Saints' Church, Sherburn-in-Elmet, Frederick Mackeown, M.A., A.C.A., younger son of Rev. W. and Mrs. Mackeown, Beausite, Rushbrooke, Co. Cork, to E. Lorraine Hayes, B.A., only daughter of Major R. Hayes, M.R.C.V.S., and Mrs. Hayes, Elmete House, Sherburn-in-Elmet.

On Leave.—Messrs. F. V. Aston and R. Coulthard, Veterinary Officers in the Colonial Service, have arrived home on leave from Kenya and Nigeria respectively.

Will.—Hancock, Mr. William Ashton, of West Drayton, veterinary surgeon (net personalty £7,686), £12,417.

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R.C.V.S. OBITUARY

Prickett, G. F. Graduated Edinburgh, December 22nd, 1891; died January 10th, 1936.

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GENERAL OBITUARY

Dr. R. G. Canti

We regret to record the death at Hampstead, at the age of 52 years, of Dr. Ronald George Canti, who was the first man in this country, and among the first in the world, to apply the art of cinematography to microscopy for the demonstration of the changes that take place in bacteria and in living tissues. "The photography of his films was of such a high standard," says *The Times*, "that he ranked also among the foremost technicians in this art. That was all the more remarkable because the technique of taking the films and making the machines for the work—all of which he designed himself—was only a means to a scientific end. That the end in itself was of the highest value is demonstrated by the fact that he was invited to show his films before the Royal Society, and to similar societies in Germany, France, and Austria, while hundreds of copies of them were purchased by scientific and learned societies wherever bacteria, tissue culture, and the problems of cancer are being studied throughout the world. Only recently, during his illness, two films that he had been working on, dealing with psittacosis and tumours of the brain, were shown to the Pathological Society of England by a colleague."

Dr. Canti was honorary scientific secretary to the British Empire Cancer Campaign; this post included that of scientific secretary to the Scientific Committee under the chairmanship of Lord Horder. His second son is a student at the Royal Veterinary College.

Dr. A. A. Gray

Dr. Albert Alexander Gray, who died on January 4th, was not only a distinguished aural surgeon, and lecturer on diseases of the ear in the University of Glasgow, before he retired from active practice a few years ago, but throughout his life he devoted himself to studying the anatomy and physiology of the ear in animals as well as in man. After relinquishing practice he took up his residence in London to pursue his investigations at the Middlesex Hospital, to which is attached the Ferens Institute, founded primarily for research in the problems of otology. For many years he was on the Council of the Section of Otology of the Royal Society of Medicine and became President of the section. The published "Proceedings" of the society's transactions are rich in Dr. Gray's contributions. He wrote and spoke with great authority, with profound knowledge, and from extensive personal experience.

He amassed a collection of specimens of the organs of hearing of vertebrates and evolved a method of demonstrating the structure of these delicate organs, which he preserved intact by a special process after dissolving out the dense bone which encloses the internal ear. Many of his specimens he presented to the Museum of the Royal College of Surgeons of England, where they are displayed, a monument of his skill and industry.

His researches into the problems of progressive deafness led him to be acknowledged by otologists as an international authority on otosclerosis, concerning which he read his last paper at a congress at Budapest last September. He was awarded the laureateship of the Lenval prize in otology in 1909 for his work on the pathology of otosclerosis. Actually up to the time of his death he was engaged in investigating tumours of the auditory nerve.

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VETERINARY SERVICES IN ABYSSINIA

Appeal for British Field Hospital

The Royal Society for the Prevention of Cruelty to Animals is organising a veterinary unit to proceed as soon as possible to Addis Ababa, there to establish a field hospital for the benefit of war animals and to carry out whatever steps are found to be practicable for the furtherance of humane work among animals already in the field and those requisitioned for future service.

It is estimated that £2,000 to £3,000 will be necessary to equip and maintain such a unit for the first 12 months. The success of the project, it is stated, rests upon the generosity of British lovers of animals, and Sir Robert Gower, Chairman of the R.S.P.C.A. Council, appeals for donations to be sent to the Chief Secretary, Royal Society for the Prevention of Cruelty to Animals, 105, Jermyn Street, London, S.W.1, the envelope being marked "Abyssinian Veterinary Relief Scheme." The fund will be administered by the Empire and Foreign Affairs Committee of the Society's Council, with the assistance of specially qualified people who will be co-opted by the Committee for this purpose.

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FOOT-AND-MOUTH DISEASE IN OXFORDSHIRE

The existence of foot-and-mouth disease was confirmed on Saturday among sheep at Adderbury, Banbury, Oxfordshire. The area subject to restrictions in the movement of animals lies in the counties of Oxford, Bucks, Northants, Warwick, and Gloucester.

VETERINARY INSPECTION IN SHROPSHIRE: COUNTY COUNCIL'S PROPOSALS CRITICISED

The *Wolverhampton Express* reports that at the close of the ordinary business at the Annual Meeting of the Wellington (Salop) branch of the National Farmers' Union a discussion took place on the action of the Salop County Council in proposing to appoint full-time veterinary inspectors for the county. The policy had been for the veterinary surgeons to attend the local farms at any time of the day and night when required and it was suggested that it was unfair that a new set of inspectors should be appointed.

Major Aston, M.R.C.V.S. (Shrewsbury), Mr. James Martin, M.R.C.V.S. (Wellington), Mr. Wilson and Mr. Allen (veterinary surgeons) also attended the meeting.

Major Aston pointed out that the veterinary surgeons believed that the continued employment of part-time veterinary inspectors would be to the advantage of the county, to agriculturalists and to the general public. They were also in agreement with carrying out the Milk and Dairies Order which had been enacted, but not yet put into force in the county.

The County Council had appointed a chief veterinary officer who, in accordance with the terms of the advertisement, "would be required to undertake and organise (acting in conjunction with the existing part-time local veterinary officers) the whole of the veterinary work of the Council in connection with the various acts and orders."

In the chief veterinary officer's report it was recommended that full-time veterinary surgeons should be appointed. That would involve considerable expenditure for work that was being very efficiently done by local veterinary surgeons.

For the Salop County Council satisfactorily to carry out the work under the Milk and Dairies Order, the Accredited Herd Scheme and the Tuberculosis Order with whole-time inspectors, at least ten would be necessary in Shropshire.

That statement was based on the fact that Cheshire employed 11 and Staffordshire nine inspectors, and that there were 30,000 more cattle in Shropshire than in Cheshire and 37,000 more than in Staffordshire; also, that in 1934 in each of four counties in England and Wales over 1,000 head of tuberculous cattle were taken out, and Shropshire was one of those four counties.

That work was done by part-time inspectors, and while in no way suggesting that there were more tuberculous cattle in Shropshire than in any other county, the work would appear to have been done satisfactorily.

In Oswestry, Shrewsbury and Wellington the markets were inspected by the local veterinary inspectors, who dealt with their clients' and strangers' cattle with impartiality and satisfaction.

There were 18 part-time inspectors and 27 private veterinary surgeons in Shropshire, working in 22 actual practices, and the influx of ten whole-time men would obviously so increase the present number as to cause a definite loss of work.

On their own initiative, said Major Aston, many farmers had cleared their herds of tuberculosis, and the work could be perfectly well done by surgeons practising in the county.

Although Staffordshire had only had a full-time staff for one year there was over £500 extra in costs. In 1934 in the West Riding of Yorkshire, with one chief veterinary officer and nine assistants, over £16,000 was spent.

The private practitioner had always merited the confidence of the farmers, and the freedom which the county had enjoyed from foot-and-mouth disease was proof of that. No better result could be obtained from full-time inspectors at a considerable cost to the ratepayers.

Mr. James Martin, M.R.C.V.S. (Wellington) also addressed the meeting and spoke strongly against the new proposals.

A discussion followed and many questions were answered. Mr. Holburn (county secretary of the N.F.U.) said the county executive were in support of the veterinary surgeons in opposition to the county scheme.

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HEREFORDSHIRE AGRICULTURAL COMMITTEE: ALDERMAN LANGFORD'S INTERVIEW WITH THE MINISTRY

At a recent meeting of the Herefordshire Agricultural Committee, Alderman Langford made a report with regard to his interview with the Ministry of Agriculture and Fisheries in connection with various diseases.

First dealing with the question of foot-and-mouth disease, he stated that during the discussion it transpired that during a period ending in 1932 the Ministry had representatives in the Argentine, whose presence had the effect of lessening infection. It was urged upon the Ministry that those representatives should be replaced, and it was more than likely, in view of the recurring outbreaks of foot-and-mouth disease in this country, they would again be represented in the Argentine, which would have some beneficial effect upon diseased carcases which had been in contact with animals being sent to this country.

The question was raised whether the infection came from Ireland, but it was stated that Ireland was as free as it possibly could be from the disease. Unfortunately, Irish cattle which had passed through two or three markets in this country had gone down with the disease, but it was maintained that this had no connection with the theory that Irish cattle brought the disease into this country. The highest authorities found it exceedingly difficult to say where the disease came from, but the Ministry were alive to the importance of keeping the country free.

In regard to swine fever, it seemed that while Herefordshire and adjoining counties had suffered considerably from it in recent years other parts of the country had escaped. The Ministry put figures before him which were astonishing and proved that in the country generally swine fever was diminishing in spite of the fact that pigs were vastly increasing in number. He pointed out the Herefordshire grievance, that although they lived close to unrestricted areas, such as Gloucestershire, Monmouthshire and the whole of Wales, the Ministry did not appear to be willing to do anything for them and attached so much importance to the 27 days' isolation that, with the support of the Livestock Committee of the National Farmers' Union, they had increased that period to 28 days. The Ministry did not think the circumstances warranted them imposing the same conditions upon other places as were imposed upon Herefordshire. At the same time, they congratulated the authorities in Herefordshire upon the safeguards they were taking in the inspection of pigs entering the markets and the National Farmers' Union were recommending that the same safeguards should be carried out generally.

In reply to comments, Alderman Langford said he felt that swine fever being the dread disease it was, farmers should be prepared to accept any reasonable restrictions imposed upon them.

The committee adopted the report of the County Diseases of Animals Committee recommending the establishment of a county laboratory for the bacteriological examination of milk and it was agreed that the County Veterinary Officer should supervise the examination of all milk samples.

It was stated that the laboratory was necessary because more and more farmers in the county were turning from beef to milk.

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ATTESTED HERDS SCHEME

The revised Attested Herds Scheme is being further considered by the Minister of Agriculture. It is understood that Mr. Elliot will require further information on the subject before he can give his approval to the revised scheme.

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WHAT IS A PRIVATE CAR?

A curious position has arisen in a case just heard at Aberdare (Glam.) when a motorist was charged with using a goods vehicle, having paid tax on it at a lower scale as a private motor-car. He was defended by the Automobile Association and the prosecution alleged that a box container placed on the rear seat of the car was an adaptation and converted the car into a goods carrying vehicle *for taxation purposes*. The Stipendiary, Mr. Bowen Davies, K.C., upheld the contention of the A.A. solicitors that the conveyance of the container did not alter the character of the vehicle and it was properly taxed as a private car. Had any other view been taken it would have meant in effect that no goods of any kind could be conveyed in a private car.

An extraordinary aspect of the matter is that it is a sequel to a case recently heard at the same Court but before a lay Bench of Magistrates when the same car with similar equipment was held to be a vehicle necessitating a goods carrying licence under the Road and Rail Traffic Act. Moreover, this decision was upheld on appeal to Quarter Sessions. The effect of these two decisions is that the car is classed as a private car for taxation but is a goods carrying vehicle for the purpose of road regulation.

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THE POISONS LIST AND POISONS RULES

The *London Gazette* of December 31st, 1935, contains an Order of the Home Secretary confirming, with modifications, the Poisons List presented to him by the Poisons Board in May, 1935, and the final text of the Poisons Rules made by him after consideration of the draft rules recommended by the Board. The Order and the Rules will now lie on the tables of both Houses of Parliament for 30 days during which the Houses have sat, and if at the end of that period neither House has resolved upon an Address praying that either Order or Rules shall be annulled, they will become permanent. No part of the Rules becomes operative until May 1st, 1936. The Poisons List is effective from the date of publication—that is, from January 1st, 1936—but as a limited number only of the sections of the Pharmacy and Poisons Act, 1933, will come into force on that day, the present Schedule of Poisons will remain the effective list of statutory poisons for practical purposes until May 1st, 1936, when the old statutory provisions controlling the sale of poisons are repealed, the Schedule of Poisons along with the rest, and the corresponding sections of the 1933 Act become operative in their place. Copies of the Poisons List, the Poisons Rules, and the form prescribed for application to be made to the local authority for registration for the sale of the substances in Part II of the Poisons List, have been placed on sale, and may be purchased from H.M. Stationery Office or through any bookseller. The price of the Poisons List is 1d., and of the Poisons Rules, 8d.

Dealing with changes in the Poisons List, *The Pharmaceutical Journal* points out that the Home Secretary has struck out of the Poisons List a number of substances, none of them except acetic acid and carbon tetrachloride widely used. The Poisons Board recommended that glacial acetic acid and carbon tetrachloride in medicines for human use should be controlled, and the Home Secretary's action affects those substances only. It is, therefore, a change of little practical importance. "The transference of some poisons from Part I to Part II of the List and the exemption of certain substances from the operation of the Rules are accompanied by a number of pin-pricks which pharmacy might reasonably have been spared. Thus, hydrochloric acid, which the Poisons Board recommended should be placed in Part I, has been transferred to Part II; similarly, lysol and solutions of lysol have been transferred to Part II and become saleable by any trader who applies to the local authority to have his name entered on the authority's list."

Commenting on the complete exemptions, the *Journal*, says that that of the solid substances containing phenols (such as the so-called lysol tablets) is noteworthy, and proceeds: "The freeing of hydrochloric acid and lysol from the fuller restrictions recommended by the Poisons Board amounts to the assumption by the Home Secretary of a heavy responsibility, in that he has ignored the advice of his experts with regard to two of the most notoriously dangerous substances in common use. Of the 886 persons who died from poisoning in 1934, approximately half were killed by these two substances!"

Concerning changes in the Rules, the observation is made that to manufacturers of animal medicines the Home Secretary has seen fit to make a surprisingly generous gesture. The Poisons Board recommended that these manufacturers should be permitted to sell medicines containing certain poisons only. The new Rules remove the limitation and so open the whole contents of the Poisons List to be distributed freely to the public by manufacturers so long as the poisons are in the form of animal medicines.

New Poison Regulations in South Africa

New regulations governing the keeping, sale or supply of poisons have been gazetted under the Medical, Dental and Pharmacy Act of 1928. They will apply throughout the Union of South Africa and South-West Africa, and came into operation on January 1st, 1936. The regulations are intended to clarify the position in regard to the sale of poisons by general and retail dealers and co-operative societies and the sale of patent, proprietary or "Dutch" medicines containing poison. The nature of the articles that may be stocked by these dealers and societies is specified, being limited to certain poisons used exclusively in agriculture or horticulture or as stock dips or for the destruction of vermin, insects, plants, fungi, and bacteria or for the preservation of wood. The dealers may also stock certain preparations containing poison to be used for veterinary purposes and registered with the Department of Agriculture and Forestry under the Fertiliser and Pest Remedies Act of 1917.

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"HENRI'S CHOICE" TO RACE AGAIN

It will be remembered that, in our issue of September 14th last, we published a case report (with skiagraphic illustration) by Mr. W. L. Cockburn, M.R.C.V.S., concerning the axial fracture

sustained by "Henri's Choice," the racehorse which fell in the Liverpool Hurdle Race on March 30th, 1935. In his report Mr. Cockburn stated that the odontoid process was broken and doubled downwards, making, with the under outline of the remainder of the axis, an angle of 70 degrees. We now learn that the animal has made a remarkable recovery, and has done so well in his training gallops that he will probably contest the Imperial Hurdles Cup, at the Grand Military meeting at Sandown Park on Saturday, March 21st.

The horse was unconscious for two days after his fall, and lay for seven weeks in the stables at Aintree with his neck in plaster of Paris.

After a long spell of veterinary treatment at Long Stanton in Cambridgeshire he returned to Owen Anthony's training establishment at Letcombe Basset, Wantage.

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THE SHIRE HORSE SHOW

There are good prospects for the Shire Horse Show at the Royal Agricultural Hall on February 4th, 5th and 6th. At a recent meeting of the Council of the Shire Horse Society it was reported that the number of entries received was the highest since 1931. The actual entry is 300, compared with 253 last year. Competition promises to be keen. Last year's champion stallion (Harboro' Goldfinder) has been entered, and will again meet his reserve (Raans Clans man). The yearling filly (Margaret of Chippinghurst), which was first in the female section last year, will again compete against her 1935 reserve (Bower June Rose). The King's Stud will again be represented. Other notable exhibitors will include the Duke of Bedford, Mr. J. Morris Belcher, Sir Gomer Berry, Mr. W. J. Cumber, the Duke of Devonshire, Messrs. James Forshaw and Sons, Mr. G. R. C. Foster, Sir Bernard Greenwell, the Liverpool Corporation, Mr. A. Thomas Loyd, Messrs. Mann, Crossman, and Paulin, Limited, Mr. E. W. Webb, and Sir Ernest Wills. The council has put forward to the members the proposal that Lord Derby should be elected President-elect.

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LIVESTOCK IN THE UNITED STATES

M. C. Bond, Extension Professor of Marketing, Cornell University, records in a recent issue of the *Cornell Veterinarian* that whereas each census period from 1850 to 1910 showed an upward trend in the total numbers of livestock on farms in the United States, since that year no continuous upward trend in numbers has been evident. The reports show a rather marked fluctuation in the number of sheep with present numbers at near the largest on record. The last two decades show a tendency towards decline in the numbers of horses, swine and cattle. Professor Bond states that with the population of the United States increasing from about 23,000,000 in 1850 to 122,000,000 in 1930 a rather marked increase in numbers of livestock would be necessary if the available supply of livestock and livestock products were to be maintained in relation to population. According to the United States census of 1930 the number of cattle per hundred persons was 52·2 compared with 75·7 in 1900, a decrease of 31 per cent.; the decline for swine was 44 per cent.; sheep 42 per cent.; horses 54 per cent., and milk cows 23 per cent.

In New York the number of horses increased from about 400,000 in 1850 to 700,000 in 1890 and then gradually decreased to 300,000 in 1930. In contrast, the number of swine showed a marked tendency towards decline from 1850 to 1930. Sheep also declined from about 3¼ million in 1850 to 600,000 in 1930. The number of dairy cows increased from less than 1,000,000 in 1850 to about 1·4 million in 1870. From 1870 to date the number of dairy cows in New York showed no long-time tendency either to increase or decrease.

The peak in the number of horses on farms in the United States was reached in 1918 with more than 20 million, this number declining steadily to less than 12 million on January 1st, 1935. The average farm price of horses in New York during the pre-war period (1910 to 1914) was 117 dollars per head. In February, 1933, the average price was 102 dollars and in April, 1935, 140 dollars. This increase of about one-third was considerably greater than the increase in the price of swine, sheep and cows, the explanation of the greater increase in the price of horses being the long period of steadily declining numbers. With the prospect of relatively high prices for horses during the next five or ten years, a marked increase may be expected in the number of colts raised.

The very low prices of swine, coupled with the short corn crop this year (1935) will probably result in a very striking rise in the next cycle, the period from peak to peak in the hog cycle being about five years. The purchasing power of sheep shows a fairly regular cycle through periods of about nine years from peak to peak, the bottom of the last cycle occurring in 1933. The unusually large number of sheep in the United States a few years ago is gradually being reduced, therefore the purchasing power of sheep may be expected to rise for several years, but there is no indication that sheep and swine will become generally as profitable forms of enterprise as dairy cows and poultry. The dairy cow cycle requires 14 to 16 years from peak to peak, the last peak occurring in 1929. The New York dairy industry is faced with relatively large numbers of dairy cows (the number at present in this State approximates 1,500,000) and relatively low prices.

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RELATIONS BETWEEN PARASITIC PROTOZOA AND THEIR HOSTS

Nature of January 4th quotes D. H. Wenrich (*Proc. Amer. Phil. Soc.*, 75 (1935)) as stating that, as the result of his observations on parasitic Protozoa, the conviction has grown that the idea of the rigidity of host-specificity has too many exceptions to make it a safe guide to the study and naming of new parasites. Every gradation can be found from early stages of facultative invasion of hosts to highly modified forms with rigid host-specificity. At one extreme are free-living species capable of living endozoically for a time when opportunity affords, for example, *Euglena gracilis* in the millipede *Spirobolus*. At the other extreme are the highly evolved gregarines and astomatous ciliates with specially adapted structures for attachment to their respective hosts and a high degree of host-specificity. Members of the flagellate genus *Giardia* are limited to vertebrates, and they also have a high degree of host-specificity, whereas the species of the nearly related genus *Hexamita* are found in a wide range of environments, including both fresh and salt water and upon or within animals belonging to many different phyla, and they show much less host-specificity. Evolution of species has been extensive in some groups and slight in others. As examples of the former the author cites the ciliate family Ophryoscolecidæ in ruminants, the polymastigote and hypermastigote flagellates in the termites

and the trypanosomes found in many different kinds of hosts. By contrast, little change has apparently taken place since the endozoic habit was established in the endozoic euglenoids and in certain of the ciliates found in sea-urchins.

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After drilling corn Mr. H. Blackburn, of Coningsby, Lincs., emptied the drill and fed some of the corn to his horses. All the animals died shortly afterwards, and it was later found that the corn had been treated with an arsenical dressing as a protection against birds.

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The year 1935 has been one of the most eventful in the history of the Red Poll breed, which, in spite of the present none too healthy outlook in the cattle industry, has made substantial progress. The wider concentration on dual-purpose cattle is reflected not only in the increased number of new herds that have been formed in different parts of the country, but also in the greatly extended use of Red Poll bulls by owners of herds of commercial dairy cattle.—*Meat Trades Journal.*

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At a special meeting of the County Diseases of Animals Committee at Hereford, a report recommending the establishment of a county laboratory for the bacteriological examination of milk was adopted and it was recommended that the County Veterinary Officer should supervise the examination of all milk samples.

Correspondence

Letters to the Editor should reach the Office not later than by the first post on Tuesday morning for insertion in following Saturday's issue.

All correspondence must bear the name and address of the contributor for publication.

The Editor does not hold himself responsible for the opinions of his correspondents.

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THE INCIDENCE OF TUBERCULOSIS

To the Editor of The Veterinary Record

Sir,—The contribution to our knowledge on the above subject given by Dr. Montgomerie and Mr. Rowlands published in last week's *Veterinary Record,* will, it is hoped, be followed by further discussion. One cannot help admiring the work of those who attempt to find a way in the elimination and prevention of this disease by the use of the tuberculin test.

The eradication of the disease from selected areas in preference to individual herds, is considered by many authorities to deserve support, but the demand for tubercle-free dairy stock must be developed with the area plan. The present incidence of tuberculosis in dairy herds is of vital importance to the profession. The recent development of a market for liquid milk and level daily deliveries of milk, has encouraged retention and purchase of older cows in herds even where the custom was to sell older cows and replace with heifers or self-supporting herds with chiefly summer milk production. This is evident where cheese and butter making were carried out; those areas where rearing of young stock and a low incidence of the disease previously existed have almost lost their indentity. This is bound to result in an increase in the price of heifers, in the average age of dairy cows and the incidence of tuberculous infections.

The usual cry is that the Government should do something about the incidence of the disease, but the industry itself is largely responsible for the present state of affairs. The most the Government can be expected to do is to offer free tuberculin testing if owners are prepared to eliminate reactors.

The development of accredited herds and clinical examinations carried out at short intervals will not reduce the incidence of the disease, but will give a measure of protection through the elimination of clinical cases.

Our profession must point out the danger of losing the source of supply of healthy stock through the increase in milk production in the rearing areas, and recommend and assist development of the attested and area eradication schemes for the supply of these animals. In this the industry must co-operate or submit to other forms of control. A more liberal offer of assistance in tuberculin testing by Local Authorities would do much in the direction of building up tubercle-free herds.

Yours faithfully,

Wm. Tweed.

County Hall,
Taunton.
January 13th, 1936.

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PROPOSED PRESENTATION TO SIR FREDERICK HOBDAY

To the Editor of The Veterinary Record

Sir,—In view of Sir Frederick Hobday's great services and the honour which he has brought to our profession, it occurred to a few veterinary surgeons, who were gathered together recently, that some permanent mark should be made in order that we might thereby signify our respect and regard for him personally and at the same time provide a record for the future.

Hobday's name will hold a revered place in the hearts of veterinary surgeons yet unborn. It is pleasing to think that those who follow us might be able to see "what manner of man was this" and so the suggestion occurred to us that we might originate a fund in order to present to Sir Frederick his portrait, executed in oils by some well-known artist. It is our hope that at some time such a portrait might grace the great hall of the London College which owes so much to his efforts. Mr. John Hassall has been approached and he has signified his willingness and pleasure to paint such a canvas.

A subscription list has therefore been opened and contributions will be accepted and acknowledged by Major Hamilton Kirk, who has consented to act as treasurer. No limit is to be placed on subscriptions, large or small, and in the event of funds exceeding the necessary sum required it is proposed to hand the surplus to Sir Frederick to use at his discretion for the benefit of impecunious students.

Yours sincerely,

H. Scott Dunn.
Hamilton Kirk.
J. McCunn.
Wm. C. Miller.

The Editor acknowledges, with thanks, the receipt of the following:—

Communications from Dr. H. P. Bayon (Cambridge), Mr. H. E. Bywater (London), and Dr. W. R. Wooldridge (London).

Report of a meeting of the North Midland Division, N.V.M.A., from Mr. H. M. Salusbury, Hon. Secretary.